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June 1957

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SECTION OF GEOLOGY AND MINERALOGY

SEDIMENTARY SUCCESSIONS IN THE PENNSYLVANIAN ALLEGHENY AND CONEMAUGH SERIES, WESTERN PENNSYLVANIA*

By C. E. Prouty

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The cyclic behavior of the Pennsylvanian rocks has been recorded in the Illinois Basin (J. M. Weller, 1930; H. R. Wanless, 1939) and in the West Virginia Basin (D. B. Reger, 1931). Weller applied the name cyclothem to the succession of nonmarine and marine rocks composing one sedimentary cycle. Wanless worked out the nature of the typical cycle and studied its lateral extent. Reger's study indicated that the West Virginia cycles were thicker and of more nonmarine composition than those of Illinois.

Several years ago I began an extended program involving participation by graduate students in the study of the Pennsylvanian sedimentary cycles in western Pennsylvania. This study, still in progress, has covered most of the Pottsville, Allegheny, and Conemaugh series and, to a smaller extent, the Monongahela series. The purpose of this paper is to bring together some of the principal findings within the Allegheny and Conemaugh, the series that best illustrate typical cyclothemic behavior in this area. Several workers have been connected with this program and are identified by references.

The Pottsville, Allegheny, Conemaugh, and Monongahela series, comprising the eastern Pennsylvanian system standard, contain but few different lithologic types throughout several hundred feet of section. Similar lithologies repeat many times throughout the various series. Distinctions between the four series are based on the relative proportions of the recurrent sandstones, shales, claystones, limestones, clays, and coals. Thus, the Pottsville has a higher percentage of sandstone, although other types are represented; the Allegheny has more shale; the Conemaugh, more well-developed marine limestones; and the Monongahela, more fresh-water limestones. Otherwise, for example, a sandstone unit from the Pottsville would be likely to show imperceptible megascopic (and perhaps microscopic and compositional) differences from a sandstone unit in the Monongahela. Criteria other than lithologic must in most cases be brought into play for correlation purposes. The most common method is the stratigraphic interval existing between one of the few key units readily recognized through some outstanding characteristics, such as thickness, color or, in a few instances, fauna.

The rocks belonging to the Allegheny series have been studied in the

*This paper, illustrated with lantern slides, was presented at a meeting of the Section on May 6, 1957.

area north of Pittsburgh (J. W. Raine, 1953; W. P. Fleck, 1953). The lower approximate half of the Allegheny series may be divided into five cycles (TABLE 1) from older to younger: the Brookville, Lower Clarion, Vanport, Lawrence, and Lower Kittanning. A standard cycle consists of nine units, in ascending order: (1) gray-brown shale; (2) gray claystone; (3) underclay; (4) coal; (5) gray shale or plastic clay; (6) black fissile shale; (7) marine limestone; (8) gray to gray-green shale; (9) and gray-brown shale. In no one cycle observed were all nine units present. However, the particular units present fall into the same position relative to other units within a given cycle. By comparing many sections, the standard section can thus be established. All units from 1 to 4 inclusive are nonmarine; all units from 5 to 9 inclusive are marine. Positive proof of a marine origin based on fauna was established only in the upper Vanport hemicycle. The occurrence of marine rocks in the other four cycles, though faunally barren, was inferred through the presence in each of units comparable to those in the definite marine unit. The nonmarine phase is established on the occurrence of land plant fossils. Thus, the lower Allegheny series, consisting of six formational names under the present generalized classification can be subdivided into over 35 units (composite section) comprising five cyclothems.

The upper Allegheny series (TABLE 2) may be similarly subdivided into more than 40 units comprising 5 cyclothems. The standard cycle typical of the upper Allegheny differs from the lower Allegheny in the presence in the former of fresh-water limestone (unit 3) and an additional shale (unit 4), and in the absence of the marine limestone (unit 7 of the lower Allegheny).

The Conemaugh series best shows the marine and nonmarine successions, a number of fossiliferous marine limestones occurring throughout the section. The Conemaugh, fully exposed in the Pittsburgh area and environs, contains the same lithologic types as the Allegheny series. The lower third of the Conemaugh (TABLE 3) may be divided into 5 cyclothems comprising nearly 45 units. The standard section (L. S. Sheafer, 1950) consists of an 11-unit cycle closely resembling the upper Allegheny standard, but with the marine limestone better developed. The middle Conemaugh (TABLE 4) has 4 cycles comprising about 35 units. The standard section (W. N. Tindell, 1950) consists of a 10-unit cycle contrasting with the lower Conemaugh in the appearance of a reddish color in some of the claystones and shales in both the nonmarine and marine phases. The upper Conemaugh (TABLE 5) is divisible into about 20 units comprising 2 cycles. The composite standard (A. J. Newmyer, 1948) consists of a 9-unit cycle. The principal difference contrasted to the middle Conemaugh is in the absence of red shales and the replacement locally of coal by carbonaceous shales. The top of the Conemaugh series, defined as the base of the Pittsburgh coal, would be at the top of the nonmarine portion of the cycle. No definite marine units are known

TABLE 1

ALLEGHENY SERIES

Lower Allegheny Series Showing the Generalized and Composite Sections, Cyclothems, and Standard Cycle

Generalized Section	*Composite section*			*Standard cycle*
	Shale, gray Shale, concretion Shale, black Clay or shale	Marine	Lower Kittanning cycle	(9) Shale, gray-brown, sandy (8) Shale, gray to gray-green, with limy iron-claystone concretions
Upper Freeport coal Upper Freeport limestone Butler sandstone Lower Freeport coal Freeport sandstone Upper Kittanning coal Middle Kittanning coal Kittanning sandstone	Coal Underclay Claystone Sandstone	Nonmarine		(7) Limestone, marine fossils
	Shale, black Clay	Marine	Lawrence cycle	(6) Shale, black, fissile (5) Shale or plastic clay, gray
Lower Kittanning coal Lawrence coal Vanport limestone Upper Clarion coal Lower Clarion coal Brookville coal	Coal Underclay Claystone Sandstone	Nonmarine		(4) Coal (3) Underclay
	Shale, gray Limestone Shale, black Shale or clay	Marine	Vanport cycle	(2) Claystone, gray; often with irregular limy nodules (1) Sandstone, gray-brown, shaly
	Coal Underclay Claystone, gray Sandstone	Nonmarine		
	Shale, sandy Claystone, gray Shale, black Shale, gray	Marine	Lower Clarion	
	Coal Underclay Claystone, gray Sandstone	Nonmarine		
	Shale, gray-brown Shale, black Clay, plastic	Marine	Brookville cycle	
	Coal Underclay Claystone Sandstone	Nonmarine		

TABLE 2

ALLEGHENY SERIES

Upper Allegheny Series Showing the Generalized and Composite Sections, Cyclothems, and Standard Cycle

Generalized section	*Composite section*			*Standard cycle*
	Shale, gray Shale, black	Marine	Upper Freeport cycle	(10) Shale, gray or brown, sandy
Upper Freeport coal Upper Freeport limestone Butler sandstone Lower Freeport coal Freeport sandstone Upper Kittanning coal Middle Kittanning coal	Coal Clay, light gray Claystone, gray Limestone and flint clay Claystone or shale Sandstone	Nonmarine		(9) Shale, gray; limestone leases or concretions (8) Shale, black, fissile (7) Shale or clay, gray (6) Coal (5) Clay, light gray (underclay)
Kittanning sandstone Lower Kittanning coal Lawrence coal Vanport limestone Upper Clarion coal Lower Clarion coal Brookville coal	Shale, olive Shale, gray concretion Shale, black Shale, gray	Marine	Lower Freeport cycle	(4) Shale or claystone, gray (3) Limestone, flint clay (2) Shale, greenish-gray or brown (1) Sandstone
	Coal Clay, gray Claystone, gray Claystone or shale Sandstone	Nonmarine		
	Shale, gray Shale, gray	Marine	Upper Kittanning cycle	
	Coal Clay Claystone, gray Shale, brown Sandstone	Nonmarine		
	Shale, gray-brown Shale, limestone lenses Shale, black Clay, light gray	Marine	Middle Kittanning cycle	
	Coal Clay, light gray Claystone, gray Shale, gray limy Shale, greenish-gray Sandstone	Nonmarine		

TABLE 3

CONEMAUGH SERIES

Lower Conemaugh Series Showing the Generalized and Composite Sections, Cyclothems, and Standard Cycle

Generalized section	*Composite section*			*Standard cycle*
Pittsburgh coal	Black shale	Marine	Pine Creek cycle	(11) Shale, brown sandy, with iron claystone concretions
Pittsburgh limestone	Limestone			
Connellsville sandstone	Black shale			
Little Clarksburg coal, clay, and limestone	Gray shale			
	Coal	Nonmarine		(10) Shale, dark gray to black, with iron claystone concretions
Morgantown sandstone	Underclay			
Wellersburg coal and clay	Claystone			
Birmingham shale and sandstone	Limestone			
Duquesne coal	Brown shale			
Grafton sandstone	Sandstone			
Ames limestone	Brown shale	Marine	Brush Creek cycle	(9) Limestone, dark, foss
Harlem coal	Black shale			
Pittsburgh redbeds	Limestone			(8) Shale, dark gray to black
Upper Saltsburg sandstone	Black shale			
Bakerstown coal	Clay or shale			(7) Shale or clay, light colored
Middle Saltsburg sandstone	Coal	Nonmarine		
Wood's Run limestone	Underclay			(6) Coal or black shale
Lower Saltsburg sandstone	Shale or claystone			
Pine Creek limestone	Limestone			(5) Underclay
Buffalo sandstone	Shale or claystone			
Brush Creek limestone	Sandstone			(4) Shale or claystone, gray or brown
Brush Creek coal	Brown shale	Marine	Upper Mahoning cycle	
Mahoning sandstone	Black shale			(3) Limestone, gray, unfoss
	Clay or shale			
Freeport coal (Allegheny)	Black shale	Nonmarine		(2) Shale and/or claystone, gray or brown, sand with nodules
	Underclay			
	Claystone			
	Limestone			
	Shale-claystone			
	Sandstone			
	Brown shale	Marine	Lower Mahoning cycle	(1) Sandstone
	Black shale			
	Gray shale			
	Coal-black shale			
	Underclay	Nonmarine		
	Shale-claystone			
	Limestone			
	Shale-claystone			
	Sandstone			
	Brown shale	Marine	Upper Freeport cycle	
	Black shale			
	Clay			
	Coal	Nonmarine		

TABLE 4

CONEMAUGH SERIES

Middle Conemaugh Series Showing the Generalized and Composite Sections, Cyclothems, and Standard Cycle

Generalized section	*Composite section*			*Standard cycle*
Pittsburgh coal	Gray-green shale	Marine	Wellersburg cycle	(10) Red shale with thin limestone lenses and/or ironstone concretions
Pittsburgh limestone	Black shale			
Connellsville sandstone	Coal	Nonmarine		
Little Clarksburg coal, clay, and limestone	Underclay			
	Gray claystone			
Morgantown sandstone	Red and olive Claystone			(9) Gray-green shale with thin limestone lenses and/or ironstone concretions
Wellersburg coal and clay	Micaceous shale			
Birmingham shale and sandstone	Red shale			
Duquesne coal	Gray-green shale	Marine	Duquesne cycle	(8) Limestone containing marine fossils
Grafton sandstone	Black shale			
Ames limestone	Coal	Nonmarine		(7) Black shale
Harlem coal	Underclay			
Pittsburgh redbeds	Limestone			(6) Coal
Upper Saltsburg sandstone	Gray claystone			
Bakerstown coal	Red and olive Claystone			(5) Underclay
Middle Saltsburg sandstone	Sandstone and/or shale			
Wood's Run limestone	Red shale		Harlem cycle	(4) Limestone dense
Lower Saltsburg sandstone	Gray-green shale	Marine		
Pine Creek limestone	Limestone			
Buffalo sandstone	Black shale			(3) Gray claystone with occasional argillaceous limestone nodules
Brush Creek limestone	Coal	Nonmarine		
Brush Creek coal	Underclay			
Mahoning sandstone	Gray claystone			
	Red and olive Claystone			
Freeport coal (Allegheny)	Sandstone and shale			
	Red shale		Bakerstown cycle	(2) Red and olive claystone with argillaceous limestone nodules especially in lower portion
	Gray-green shale	Marine		
	Black shale			
	Coal	Nonmarine		
	Underclay			
	Gray claystone			(1) Gray-green, micaceous sandstone and/or sandy, micaceous shale

TABLE 5

CONEMAUGH SERIES

Upper Conemaugh Series Showing the Generalized and Composite Sections, Cyclothems, and Standard Cycle

Generalized section	*Composite section*			*Standard cycle*
	Shale, sandy, limestone concretions			(9) Shale, gray claystone or limestone concretions
Pittsburgh coal	Shale, black, sheety			
Pittsburgh limestone	Shale, gray, clay			
Connellsville sandstone	Shale, sheety			
Little Clarksburg coal, clay, and limestone	Coal		Pittsburgh coal cycle	(8) Shale, black, locally coaly
Morgantown sandstone	Underclay			
Wellersburg coal, clay	Limestone, buff-gray	Nonmarine		(7) Limestone, often brecciated
Birmingham shale and sandstone	Shale			
Duquesne coal	Sandstone			
Grafton sandstone	Shale, gray			
Ames limestone	Shale, black	Marine		(6) Shale, black, coaly
Harlem coal	Limestone			
Pittsburgh redbeds	Shale, black, coaly		Clarksburg cycle	(5) Underclay
Upper Saltsburg sandstone	Underclay			
Bakerstown coal	Shale, clay	Nonmarine		(4) Shale, gray, few limestone nodules
Middle Saltsburg sandstone	Limestone			
Wood's Run limestone	Shale			(3) Limestone
Lower Saltsburg sandstone	Sandstone			
Pine Creek limestone				(2) Shale, gray, limestone concretions
Buffalo sandstone				
Brush Creek limestone				(1) Sandstone, locally shaly
Brush Creek coal				
Mahoning sandstone				
Freeport coal (Allegheny)				

in the Monongahela series, and the rhythmic development of the various lithologies is largely interrupted by thick transitional sandstones, sandy shales, and fresh-water limestones.

The recurrence of numerous thin lithologic units has created considerable difficulties in Pennsylvanian nomenclature and classification. In the past only the prominent and more readily recognized units have been assigned formational names. Unlike most other parts of the geologic column, many known gaps exist between designated formational units. Few of the thinner units are regionally persistent, although coal beds and marine units are commonly exceptions. Thus, the units are more like members than formations in lateral distribution. The revision of the nomenclature according to the cyclothem concept appears to be the most logical procedure, with individual cyclothems assuming formational rank and presently named formational units, taking on member status. Numerous thin lithic units would remain unnamed, but it is not thought feasible to assign hundreds of names to such ephemeral units, under any classification. If existing formations were lowered to member status, they would represent only the important members of formations (cyclothems), an arrangement more compatible with nomenclatural practices.

In summary, the following observations concerning these sedimentary successions may be made:

(1) The repetition of similar type rocks occurs many times.

(2) Definite faunal evidence for both marine and nonmarine sequences occurs in some sections; others may be assigned marine or nonmarine status by comparison to lithologically similar fossiliferous units.

(3) The average thickness of the western Pennsylvania cyclothem (approximately 35 ft.) exceeds the average of the Illinois cycle, but is less than the West Virginia cycle.

(4) The marine phase is not as complete as in Illinois cycles; the latter are more fossiliferous and contain two, rather than one, marine limestones.

(5) The number, arrangement, and lithology of the units comprising the average western Pennsylvania cycle compare rather closely with the same characteristics of Illinois cycles.

(6) The thicker and more "continental" nature of the western Pennsylvania cycles as compared to that of Illinois cycles infers the eastward source of the clastic sediments.

(7) A study of the sedimentary environments of the Pennsylvanian sediments reveals considerable data concerning the paleogeographic history of this area.

(8) A study of Pennsylvanian sediments along the lines indicated in TABLES 1 through 5 is considered the best procedure for classifying sediments of this age.

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SECTION OF BIOLOGY

STUDIES ON MAMMALIAN MAST CELLS*

By Jacques Padawer**

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Introduction

In a thorough series of experiments, culminating in 1939 with the publication of a book on heparin,[1] the Scandinavian school established the concept that mast cells secrete a sulfonated acid mucopolysaccharide having remarkable anticoagulant activity. At the turn of the century this compound had been extracted from liver, and mast cells had been described by Paul Ehrlich a few years before, but it was not until 1937 that a relationship was established between the two. So decisive were the arguments presented that it was generally assumed that the problem of mast cell function was settled, a belief apparently still held by many today. At present, primarily through the work of J. F. Riley linking mast cells with histamine, a new effort is being exerted to investigate the "solved" problem of their physiological significance.

Much has been written about mast cells, and the reader is referred to the reviews of Arvy,[2] Asboe-Hansen,[3] Baker and Abrams,[4] Michels,[5] Riley[6-8] and Speirs.[9] In this paper, we shall not attempt to cover the extensive literature of the subject, but we shall pay particular attention to some aspects of mast cell physiology with which we have had first-hand experience.

What Is a Mast Cell?

The very nature of the mast cell is apparently still a matter of debate. Indeed, a clear-cut distinction often is not made between blood basophilic leukocytes ("mast leukocytes") and "tissue" mast cells ("tissue basophils"), and any such a distinction may yet prove to be untenable. One argument suggests that mast cells are but polysaccharide-laden macrophages.[10] Many of these contradictions are more apparent than real, however, and much of the disagreement can be resolved by careful evaluation of experimental data. For instance, the work of Higginbotham and Dougherty[10] establishes clearly the fact that cells staining metachromatically can be evoked by the injection of various polysaccharides and their complexes with basic substances ("micellophagosis"). This process, leading to the formation of cells which Higginbotham and Dougherty termed quasi

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mast cells, is inferred to be the one operative physiologically in the generation of the tissue mast cell.[10] The experimental design employed by these investigators is essentially that used by von Schürer,[11] who showed a striking increase in "mast cells" following the injection of heparin. Such "mast cells," however, can be digested away by Jaques' heparinase while native mast cells retain their staining characteristics.[12] Fibroblasts will ingest native mast cell granules as well,[13] and presumably do so following mast cell damage *in vivo*. From the work of Higginbotham and Dougherty,[13,14] it is therefore apparent that, to paraphrase a well-established saying, all is not mast cell that stains metachromatically. The reported clumping of mast cell granules following adrenal corticosteroid administration[3] and other treatments might well require re-evaluation in the light of such findings.

Tissue mast cells have a round vesicular nucleus, while basophilic leukocytes display a polymorphic nucleus typical of leukocytes. All stages of leukocytic differentiation are demonstrable in bone marrow for basophilic white blood cells.[5] In the rat, mast cells are numerous, while basophilic leukocytes are rare. The opposite apparently holds for the rabbit. Thus, there are good grounds for considering "tissue" mast cells as distinct entities and, until more pertinent data accumulate, it is advantageous to bypass the mast leukocyte-tissue basophil controversy. After all, this is only a matter of semantics, and it is of little import as long as it is clearly recognized as such. It is hoped that this attitude will not encourage researchers to neglect basophilic leukocytes and that they will keep on the alert for information that might help resolve the present uncertainty. In this exposition, I shall consider mast elements as exclusively of the tissue type and different from "quasi" mast cells.

Origin of Mast Cells

Having defined my object, I am still faced with the problem of the genesis of these cells. It is commonly stated that mast cells occur in tissues in particularly close apposition to blood vascular channels, a relationship interpreted by some as due to the fact that mast cells arise from precursors in the adventitia of blood vessels and gradually move away into avascular areas of connective tissues.[15,16] Others hold that mast elements arise in avascular areas of connective tissues from fibroblastic precursors and move toward the blood vessels. So far, it is impossible to identify mast cell precursors in the absence of their metachromatic cytoplasmic granules. It would be important to establish beyond question whether differentiated mast cells normally undergo mitosis. There is no doubt that binucleate mast elements are occasionally encountered in man[17] and rat[18] (FIGURE 1)*. Both mitosis and amitosis

*Smith and Lewis,[19] reporting on mast cells of the rat and hamster, note finding binucleate cells. It is not clear from their report whether these occurred in both species or in only one or the other.

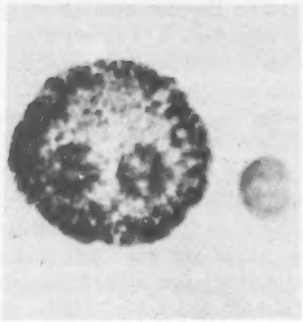

FIGURE 1. Binucleated mast cell from rat peritoneal fluid. Hematoxylin and toluidine blue stain.

have been reported in tissue cultures of rat[20] and human[21] material, as well as *in vivo* in human *urticaria pigmentosa.*[22] Mitosis has been noted in rats following treatment with histamine[23] or with 48/80[24] (p-methoxyphenethyl methylamine-formaldehyde polydisperse condensation product). Cramer and Simpson[25] did not find mast cell mitoses in skin carcinogenesis. Fawcett[16] emphasizes the occurrence of mast cells in pairs following 48/80 administration as suggesting cell division either by mitosis or amitosis. He apparently has not visualized mitoses. For the most part, these may be abnormal situations, and they may not represent the physiological picture. In view of the findings of Higginbotham and Dougherty already discussed above,[10,13,14] reports of mitosis in mast cells following noxious agents such as 48/80[24] or histamine[23] must be interpreted very cautiously. In our own studies involving an appreciable number of tissue spreads or mesenteries and very numerous samples of peritoneal fluid, we have yet to see a mature mast cell undergoing mitosis in an intact animal. In rat peritoneal fluid samples, all the binucleate cells we have visualized, either in smears or in the hemacytometer, have been very nearly spherical, with no suggestion of impending cytoplasmic cleavage. Colchicine injections have failed to demonstrate the occurrence of mitosis in mast cells of the rat.[26] Similarly, no evidence for mitosis was found following treatment with various purine and pyrimidine analogues, urethane, or podophyllotoxin – all known to have karyostatic activity (see below).

From the data available, it therefore appears unlikely that well-differentiated mast cells can multiply mitotically.

Distribution of Mast Cells

The distribution of mast elements along blood vascular channels has perhaps been overemphasized. Indeed, in subcutaneous tissue spreads

and in mesenteric preparations, they often line small nerves (FIGURES 2, 3, and 4) and lymph channels similarly. It might be that some factor leads to their accumulation at certain tissue interfaces. As already pointed out by Riley and West,[27] mast cells are very numerous in the liver capsule of the ox, pig, and dog; that is, in tissues that are almost avascular.

The question of mast cell distribution is closely related to that of their motility. Although numerous authors have referred to mast cell "migration" into particular tissue sites, little evidence has been offered to show whether this involves an active process on the part of the cell under *in vivo* conditions. In tissues such as mesenteries, there may well

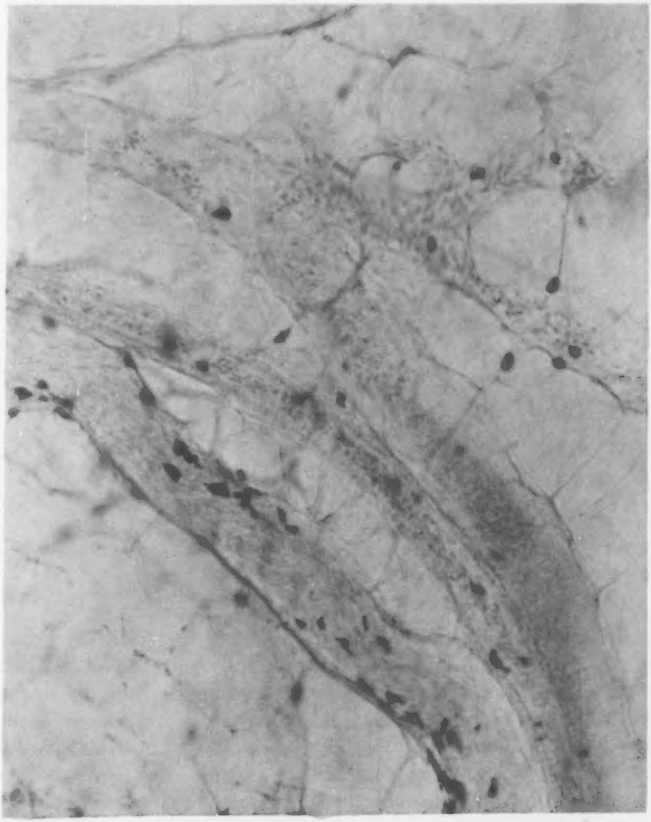

FIGURE 2. Mesenteric spread from chicken showing mast cells in proximity to a nerve trunk (left). Only an occasional mast element is found associated with nearby venous (middle) and arterial (right) channels.

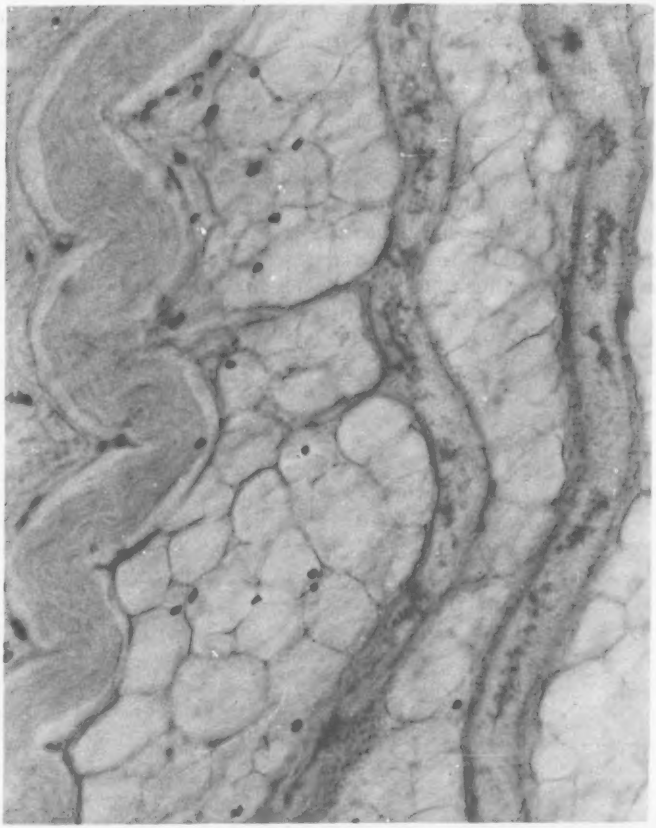

FIGURE 3. Subcutaneous connective tissue spread from chicken showing many mast cells along a nerve trunk (bottom). Only a few mast elements are seen near an arteriole (center) and a venule (top).

be more shedding of mast cells from nonvascular sites than from the immediate vicinity of blood vessels; such a differential in what has been shown to be an actual process[28] would lead to greater mast cell concentrations along blood vessel areas and could easily suggest an apparent migratory trend. I have observed mast cells of rat peritoneal fluid in a microscope incubator and have not seen any evidence of ameboid activity over a period of 18 hours, even though other elements (eosinophils and monocytic cells) were actively motile during the initial hours of observation. Mast cells derived from mastocytoma tissue[20] or skin[21] exhibit motility when grown in tissue cultures.

Staining Properties

Following Ehrlich's discovery of mast cells and their metachromatic properties, numerous investigators studied their phylogenetic occurrence and reported mast cells to be absent from various species, including fish and birds. It remained for Michels[5] to demonstrate the extreme solubility of the metachromatically staining mast cell material in *Cyprinus carpio*. This solubility was assumed to be a factor in all species and special fixative solutions preconized. This phenomenon has proved to be poorly understood. Indeed, whereas the use of aqueous solutions may lead to reports of few or no mast cells in species such as fish and birds, where

FIGURE 4. Nerve trunk of chicken mesentery. Numerous mast cells are visualized between the fibers. This nerve trunk was appreciably larger than those seen in FIGURES 2 and 3.

they are extremely water soluble, such procedures can be followed with impunity with many mammalian species. Thus, Montagna *et al.*[29] showed that human mast cells can withstand extended manipulations in aqueous media. A brief recapitulation of solubility properties of mast cells in various species I have studied is given in TABLE 1. Solubility is a function of the type of fixative used, even when it is aqueous, as exemplified by 4 per cent lead subacetate solution often used for the study of mast cells. In my hands, aqueous lead subacetate has yielded erratic results, often leading to unsatisfactory fixation and to extensive precipitate deposition within the tissues.

TABLE 1

SOLUBILITY OF MAST CELL GRANULES IN AQUEOUS MEDIA

Species	*Mast cell source*	*Solubility in water*
Rat	Peritoneal fluid	–
	Mesentery, subcutaneous fascia	–
Mouse	Peritoneal fluid	–
Golden hamster	Peritoneal fluid	–
Chinese hamster	Peritoneal fluid	–
	Cheek pouch	–
Chicken	Mesentery, subcutaneous fascia	++++
Pigeon	Mesentery	++++
Bat	Peritoneal fluid	–
	Wing membrane	–

Experiments with 48/80 have shown that it can abolish the metachromatic properties of mast cells in air-dried or methanol-fixed smears of rat peritoneal fluid.[30] This is a progressive effect: the granules still stain orthochromatically if the concentration of 48/80 is low or if the treatment is very brief. With greater concentrations, staining is inhibited altogether, no matter how brief the exposure. Other histamine liberators have been observed to discharge toluidine blue from supravitally stained mast cells of the rat.[31] Since compound 48/80 is a basic substance, other compounds tested as controls proved protamine to be even more active in this respect (on a weight basis) and NaOH most effective. The loss of stainability is not reversible by the use of acetic or hydrochloric acid at various concentrations and is probably due to dissolution of mast cell substance into the medium. This behavior is reminiscent of that described for cartilage by Benditt and French,[32,33] who found that histone blocked metachromatic staining reversibly, while N/10 NaOH caused the release of hexosamine into the medium irreversibly.

Mast cell granule solubility is therefore a complex problem exhibiting great variability from species to species. For each case at hand, the techniques employed must be carefully controlled, and findings that suggest loss of staining or even lack of mast cells require careful evaluation.

Staining reactions other than with metachromatic dyes have been discussed by Compton.[34] Techniques for the demonstration of lipids have interested us because of the possible significance to atherogenetic processes in which mast cells might play a part. Berg,[35] using benzpyrene-caffeine solution, showed that mast cells contain fat. Fadem[17] reported that human mast cells do not stain with the Jackson acetic-carbol-Sudan III technique, but Montagna and Noback[36] succeeded with a Sudan black *B* method following Baker's formol fixation. Wislocki and Dempsey[37] had also demonstrated some Sudan black *B* staining in human bone marrow material. Mast cells in tissue culture stain with Scharlach *R* when they are dying.[21] Here again, the procedures followed can determine the data obtainable. In rat peritoneal fluid smears, mast cells do not stain with Sudan black *B* in 70 per cent ethanol; eosinophilic leukocytes do stain (FIGURE 5). Following fixation with Baker's formol, staining with Sudan black *B* in propylene glycol[38] leads to the opposite results (FIGURE 6). Obviously, it is not sufficient to report staining with Sudan black *B*, but all other technical details must be supplied. It should be noted that staining with Sudan black *B* does not necessarily mean fat is present, since much material can subsequently withstand solvent treatment as well as mounting in resin. We have tried to avoid nonspecific staining by using acetylated Sudan black *B*, but this modification has yielded erratic results.

Separation of Mast Cells

Staining reactions are inadequate to reveal the chemical composition of cells. They can best be complemented by chemical procedures. Early attempts to deduce mast cell composition consisted of assaying tissues, no matter how heterogeneous, for a given substance and then correlating the data with the numbers of cells present. *Per se*, this approach is not justifiable, since an infinite number of substances could be tested for and only two possible situations prevail: correlation or no correlation. When additional data are available, such as metachromasia of mast cells *in situ*[39] or their damage following peripheral administration of various histamine liberators,[30] the suggestive correlation may well be reliable as, indeed, it proved to be with both heparin and histamine. This approach cannot constitute proof, however, and indeed is adequate only for qualitative estimation – and this only for substances present in large concentrations. Mastocytomas, approximating pure cellular populations, have yielded appreciable amounts of both heparin and histamine. Mastocytomas are still relatively heterogeneous tissues, however; they contain

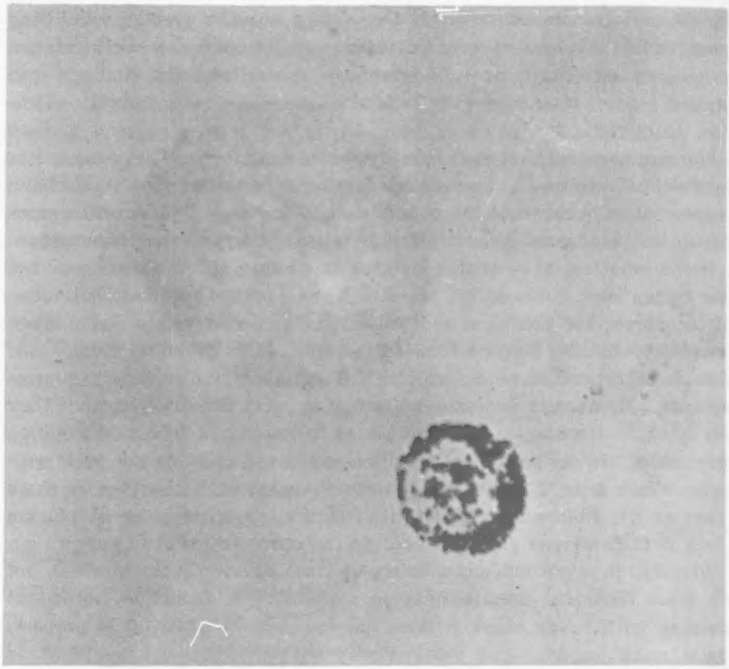

FIGURE 5. Peritoneal fluid smear from the rat air dried and then stained for 1 hour with Sudan black *B* saturated in 70 per cent alcohol. The small cell, an eosinophil, displays well-stained granules and a "doughnut" nucleus (unstained). A large mast cell nearby did not stain. Mounted in Harleco resin.

various other cellular types such as erythrocytes, leukocytes, vascular endothelium, fibroblasts, ground substance, and various connective tissue fibers. Purer populations of mast cells, in the order of 80 to 100 per cent purity and free of intercellular matrix, can be obtained in suitable species. In rats, mice, and hamsters both peritoneal[40] and pleural fluids contain mast cells. Their numbers vary not only among species, but also among strains of a given species[40] and with the age of the animal.[41] There are about 3½ per cent mast cells in the peritoneal fluid of a 200 gm. rat of the Long-Evans strain, but about 7 to 12 per cent in samples from the Sprague-Dawley strain. A method for separating the mast cells from the other elements of rat peritoneal fluid has been described.[42] Glick and his colleagues[43] have adapted this method to the continuous gradient technique; their use of heparin, while inconsequential for some purposes, may interfere with others. Benditt and others[44] also modified my method. I have now modified my original technique as follows. Adult rats (180 to

300 gm.) are injected intraperitoneally (under light ether anesthesia) with 20 to 25 ml. of a solution (solution *A*) composed of:

Tetrasodium EDTA (Bersworth)	0.00001	gm.
NaCL	0.85	gm.
Gelatin	0.01	gm.
Distilled water	100.00	ml.

This solution is brought to room temperature before injection. The animal is allowed to regain consciousness and to move about for 5 min. and is then decapitated. The peritoneal cavity is then exposed by laparotomy along the mid-ventral line, and as much fluid as possible is collected with a medicine dropper and transferred to a tube kept at ice-bath temperature. Following 8 min. of centrifugation at approximately 1000 RPM (International centrifuge, size 2, Model *V*, with head No. 250) the supernate is decanted, the cells resuspended and washed twice with ice-cold

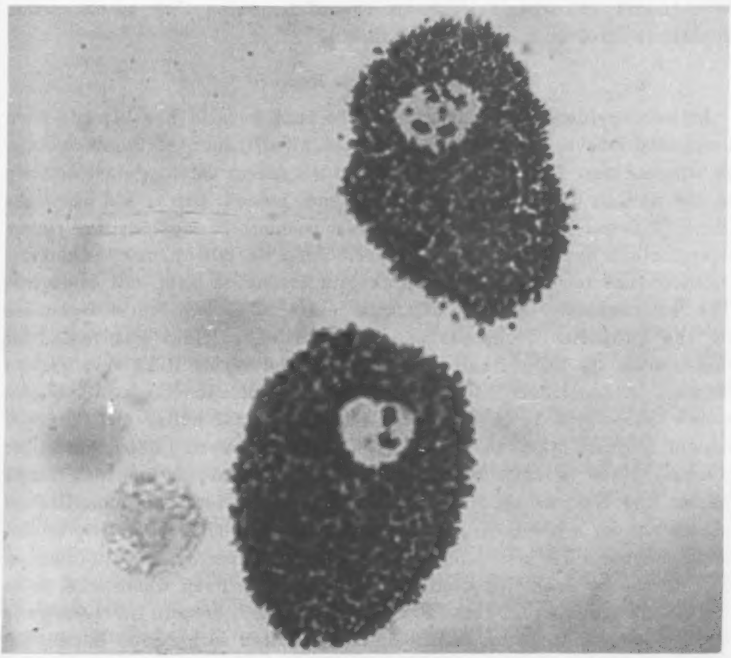

FIGURE 6. Companion slide to FIGURE 5, but air dried and fixed in Baker's formol before staining for 2 hr. with Sudan black B in propylene glycol. Two large mast cells are well-stained, while an eosinophil (lower left) has not taken up any dye. Mounted in Harleco resin.

solution *A*, and finally resuspended in approximately 3 ml. of this same medium. One half the cell suspension is now drawn up into a Van Allen tube. High density sucrose solution (stock sucrose as described elsewhere[42]), diluted just before use with 0.00001 per cent Na_4EDTA is then drawn *slowly* up into the Van Allen tube until the interface reaches the belly of the bulb and thus its maximal area. The clip is then fastened, taking care to avoid mixing the two layers. Proceed similarly with the remainder of the cell suspension. These two tubes are balanced in 50 cc. centrifuge cups and spun for 6 min. at 100 *g* (700 RPM). Allow the centrifuge to come to rest without braking, but with the cover opened. The clips are removed and the lower layer allowed to drain by gravity into a tube, taking care to stop before contaminating the yield with interface cells. Under proper side lighting conditions, the process can be carefully controlled with the naked eye. The mast cells, now in sucrose solution, are diluted with an equal volume of ice-cold solution *A* and centrifuged for 16 min. at 700 RPM. After decantation, the cells are washed twice with saline and are now ready for chemical analysis or biochemical characterization. It should be noted that no exogenous heparin is involved in this procedure.

Mast Cells and Heparin

Indirect evidence that mast cells contain heparin has already been discussed above, but the physiological significance of those findings is still unclear. Paff and Bloom[20] could not detect anticoagulant activity in the medium of their tissue cultures and, indeed, Oliver and his associates[45] found that dogs with kilogram amounts of mastocytoma tissue nevertheless had normal clotting time. After 48/80 treatment in the rat, clotting time remains unchanged despite extensive mast cell damage.[46] The anticoagulant activity of tissue extracts is apparently dependent on the extraction technique used.[47] Peritoneal fluid was tested for heparinoids by Mergenthaler and Paff,[28] who report it is very rich in heparin, although the "fluid" they analyzed includes the cellular elements suspended in it (including numerous mast cells) and therefore cannot indicate whether free heparinoids are present. If peritoneal (or pleural) fluid is collected so as to avoid contamination with tissue juices and then allowed to stand at room temperature in nonsiliconed glassware for a few hours, it is possible to determine its ability to clot spontaneously. We have done this for various laboratory animals (TABLE 2). It can be seen that clotting need not occur even where mast cells are absent (guinea pig, cat). Rat peritoneal fluid, despite its richness in mast elements, will clot readily if contaminated with tissue juices, but will not clot merely upon the addition of protamine. It is probable that fibrinogen and prothrombin are available since clotting can be induced by tissue juices or rat serum. Whether mast cell polysaccharide is actually released and physiologically available to interfere with coagula-

tion processes is therefore still unresolved. It appears more likely that mast cell heparinoids will prove to be involved in fat transport or metabolism, as suggested first by Hahn's demonstration of the lipemia-clearing activity of heparin.[48] By direct extraction of isolated mast cells,[49] it has been shown that they contain about 30 μg./mm.^{3} of heparin. If we assume a cell density of approximately unity, this represents a value of 30 μg./mg., or about 3 per cent of their wet weight.

TABLE 2

MAST CELLS AND CLOTTING ABILITY OF PERITONEAL FLUID IN LABORATORY ANIMALS

Species	*Mast cell count in fluid*	*Clotting ability*
Rat	High	–
Mouse	Appreciable	–
Golden hamster	Appreciable to high	–
Chinese hamster	Appreciable	–
Guinea pig	0	–
Rabbit	0	++++
Cat	0	–

Mast Cells and Histamine

It has been firmly established by Riley and his co-workers[31,50,51] and by others[44,49,52] that mast cells are rich in histamine. By direct analysis of relatively pure rat mast cell populations[49] their histamine content has been determined to be 10 μg./mm.^{3} of cells. If we again assume a tissue density of about unity as a first approximation, this high histamine value (equivalent to 10 μg./mg. wet weight or approximately 1 per cent) is compatible with Riley's findings for tissues with varying mast cell populations, but emphasizes the fact either that mast cells represent a small proportion of mastocytoma tissue (950 μg. histamine/g, or 0.1 per cent) despite its appearance under the microscope, or perhaps that tumor mast cells are very different from those in peritoneal fluid.

Mast cells are damaged by histamine liberators in what may be a reversible manner.[16,50] The action of these liberators on mast cell cytology is prevented by antihistaminics, although the discharge of histamine remains unaffected.[31] Antihistaminics apparently also protect mast cells against X irradiation.[53] A generalized effect following histamine liberator treatment is difficult to obtain[54] unless excessive doses are administered, preferably intravenously. Large amounts of histamine are bound by tissues other than mast cells,[55] especially stomach wall.[56] Riley

has suggested that stomach mucins, which are sulfated mucopolysaccharides, may bind histamine.[56] Reports linking mast cells to ulcer formation following 48/80 treatment[57] must therefore be interpreted with care. Few mast elements are apparently present in stomach mucosa.[56]

Rat peritoneal fluid mast cells contain a soluble histidine decarboxylase;[58] they bind the resulting histamine[58] and therefore one need not postulate that they also bind tissue histamine (and other amines), as suggested by Higginbotham and Dougherty,[10] a possibility that nevertheless cannot be ruled out at present. It has not yet been established whether mast cell histamine is present as a heparin complex or bound in some other way, although the question has aroused much interest.[55] Hedbom and Snellman[59] have suggested that the histamine of mast cells from ox liver capsule is associated with the granules while the heparin is in the surrounding cytoplasm. A similar suggestion by Julén and her associates,[60] based on metachromasia in frozen-dried material, has led to the isolation of a cytoplasmic lipoprotein-heparin complex.[61] Köksal,[62] on the other hand, has extracted heparin from mast cell granules. These granules swell during 48/80 treatment *in vitro*,[63] an observation compatible with the above findings.

Mast Cells and Serotonin

Direct analysis of mast cells isolated from rat peritoneal fluid[44] shows that they contain approximately 0.7 μg. of serotonin (5-hydroxy tryptamine) per cubic millimeter of cells, or somewhat under 0.1 per cent of their wet weight. The significance of this finding is not clear at the present time. It may eventually afford a clue to the presence of mast cells along nerve fibers. Since mast cells are damaged by histamine liberators, presumably because they contain histamine, we have looked for analogous cytological changes in mast cells following treatment with reserpine, a serotonin liberator. Under the conditions of our experiments, reserpine (either as a free base or as its more soluble phosphate) has proved without effect even at very high dosages (10 mg./100 gm. of body weight).

Mast Cells and Atherosclerosis

Hahn's findings that heparin leads to rapid clearing of blood lipemia,[48] together with the demonstration that large lipoprotein molecules (responsible for the lipemia) are a conspicuous feature of blood from atherosclerotic patients,[64] suggest that mast cells somehow might be related to some aspect of atherogenesis. Enumeration of mast cells in human aorta and myocardium[65,66] has revealed that they are less numerous in men than in women. Mast cells are present in fewer numbers among atherosclerotics than nonatherosclerotics. Constantinides[67] has suggested that the resistance of rats as compared to the susceptibility of rabbits to dietary atherogenesis might be due to a difference in mast cell numbers, the former being well supplied with mast elements while the

latter are not. We have studied chicken tissues and, with standard staining techniques (aqueous toluidine blue or Bensley's pinacyanole-erythrosinate), very few mast cells are indeed evident. Under carefully controlled conditions, however, very numerous mast cells can be demonstrated in mesenteries and in loose connective tissues of the chicken, where they are as numerous, in fact, as those we have seen in the rat. Inasmuch as the chicken is very susceptible to dietary induction of atherosclerotic lesions, mast cells *per se* are probably not the determining factor, but it may well be that their relative solubility should be taken into account. It is not clear whether differences in mast cell numbers in aorta or myocardium tissues from atherosclerotic versus nonatherosclerotic patients[65,66] are the result of atherosclerosis or its cause. When entertaining this type of correlative studies, it is well to remember that the pathology of atherogenesis in man is not in all respects identical to that elicited in birds or rodents by dietary modifications.

Aging apparently affects mast cell numbers differently in various tissues. In men and women, they occurred in equal numbers in both aorta and myocardium from young and old nonatherosclerotic patients.[65,66] Mast cells in the walls of various veins and arteries of human material were enumerated by Sundberg.[68] Helström and Holmgren[69] found that mast cells of human skin and heart decrease with age. In rat peritoneal fluid, where quantitation is more easily achieved, mast cell numbers increase as a function of aging.[41,70] Preliminary investigations suggest that the gelation state of mast cell cytoplasm is age-dependent.[71]

Mast Cells and Antimitotic Agents

Colchicine leads to polymorphism in mast cells,[18,26,72] a change accompanied by a characteristic displacement of the nucleus to the periphery of the cell.[18,26] No nuclear aberrations are evident. Colchicine does not affect the stainability of mast cells with toluidine blue.[30] Under suitable conditions, it affects the cytology of mast cells *in vitro* in a manner identical to its *in vivo* effect and therefore it probably acts directly.[73] Podophyllotoxin is similarly active.[73] (FIGURE 7).

The colchicine effect has been studied in more detail, and we have established that all antimitotic substances do not act similarly. Thus, many antimetabolites, some of which are listed in TABLE 3, do not affect mast cells visibly, suggesting that the morphological changes seen following colchicine treatment are independent of the mitotic cycle. Tumor-damaging activity and antimitotic activity have been used to assay various colchicine[74-77] and podophyllotoxin[78,79] analogues. These assays are time-consuming, require appreciable amounts of material and, in the case of tissue cultures, are perhaps difficult to quantitate. The mast cell response to these agents is so exquisitely sensitive that it has been possible to devise a mast cell assay that we carry out as follows.

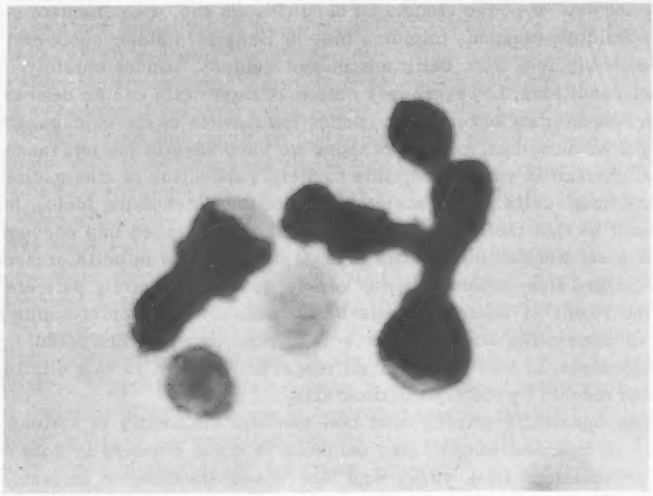

FIGURE 7. Mast cells from rat peritoneal fluid 3 hr. after subcutaneous podophyllotoxin injection as seen in hemacytometer. Note their pronounced departure from the normal spherical shape. Type of branching seen here is obtained with amounts above minimum effective dose (MED). With MED, mast cells look like the one on the left. The nucleus, at the periphery of the cell, appears essentially normal. This picture is identical to that obtained after colchicine or any of the active derivatives of TABLE 4.

TABLE 3
EFFECT OF ANTIMETABOLITES

Compound	*Antimitotic or tumor-damaging activity*	*Mast cell effect*
A-Methopterin*	+	–
2,4-Diamino-5-(3′,4′-dichlorophenyl)-6-ethyl pyrimidine (50-276)**	+	–
2,6-Diamino purine**	+	–
8-Azaguanine**	+	–

*Methotrexate obtained through the courtesy of Thomas E. Jukes, Lederle Laboratories, Pearl River, N.Y.

**Courtesy of Samuel Bieber, Wellcome Research Laboratories, Tuckahoe, N.Y.

The substance under investigation is dissolved in an appropriate solvent (water, saline, dilute alchohol, or propylene glycol) and injected subcutaneously in rats (90 to 120 gm.) fed and watered *ad libitum*. Three hours later, the animal is exsanguinated under light ether anesthesia and a peritoneal fluid sample obtained as described elsewhere.[80] The percentage of mast cells showing evidence of damage (both polymorphism and nuclear displacement) is assessed in the hemacytometer for various dosages of the drug; effective dosage is set arbitrarily at the amount that results in damage to 70 per cent or more of the mast cell population. It is important that no anesthetic be used prior to sacrifice and that the animals be all of the same age and sex. We have found rats of the Sprague-Dawley strain* to be best; these apparently have more peritoneal fluid mast cells (8 to 12 per cent) than animals of the Long-Evans type (about 3 per cent).

Using this assay, we have tested a number of colchicine and podophyllotoxin derivatives, some of which have been grouped in TABLE 4 together with a histamine liberator (48/80), a serotonin liberator (reserpine) and aminoguanidine, shown by Schayer and his associates[81] to inhibit histamine metabolism. Only qualitative notations as to activity are indicated in TABLE 4. It can be seen that the characteristic aberrations resulting from colchicine treatment do not obtain after 48/80, reserpine, or aminoguanidine. On the other hand, many analogues or derivatives of either colchicine or podophyllotoxin are active in this respect. Of the compounds I have tested so far, activity assayed by the mast cell test has paralleled the activity of the substance as assayed by tumor-damaging activity[76-78] and mitotic inhibition;[74,75,79] by these criteria, minimum effective doses (MED) have been approximately the same, and so have relative activities (MED ratios). The success of desacetylmethyl colchicine (Colcemid) in the control of certain leukemias reported by Moeschlin and his colleagues[82] shows that modification of the colchicine molecule might yield compounds of value in human therapeutics. The ease and rapidity of the mast cell assay should help in the evaluation of available compounds.

Since colchicine is effective *in vitro* under suitable conditions, it should be possible to evaluate compounds even when only minute amounts are available. We are in the process of perfecting such an *in vitro* assay with the hope that relative activity may be determined unhampered by *in vivo* variables, such as rates of absorption from injection site and rate of excretion or metabolism by other tissues.

The parallelism between antimitotic and mast cell-damaging activity is of particular interest, since it might give us a clue as to how colchicine acts at the biochemical level; it might also, perhaps, afford more insight into the mechanisms of mitosis. It will be recalled that differentiated mast cells most probably do not undergo mitosis (see above).

*Procured from the Holzman Company, Madison, Wis.

TABLE 4

EFFECT OF COLCHICINE, PODOPHYLLOTOXIN DERIVATIVES AND OTHER COMPOUNDS

Compound	*Antimitotic or tumor-damaging activity*	*Mast cell effect*
Colchicine	+	+
Colchiceine	–	–
Desacetylmethyl colchicine*	+	+
Trimethyl colchicinic acid methyl ether d-tartrate*	+	+
Podophyllotoxin**	+	+
β-peltatins**	+	+
Urethane	+	–
Reserpine*	+	–
48/80†	?	–
Aminoguanidine	?	–

*Colcemid and Serpasil courtesy of Robert Gaunt, CIBA Pharmaceutical Products, Inc., Summit, N.J.

**Courtesy of George Motoasca, S.B. Penick and Co., New York, N.Y.

†Courtesy of Edwin J. deBeer, Wellcome Research Laboratories, Tuckahoe, N.Y.

That colchicine affects some basic aspects of mast cell biochemistry appears likely from preliminary experiments. Whatever these effects are, they ultimately must affect sol-gel equilibria within the cell because the morphological aberrations seen suggest a shift to greater gelation and can be reversed by the application of high hydrostatic pressures.[83] From the cytoplasmic sol-gel analysis of mast cells in the pressure centrifuge,[71] there is no reason to believe that they differ in any major respect from other cells studied with these techniques.[84-87]

Mast Cells and Hormonal Agents

Only the pituitary-adrenal axis and growth hormone will be considered in this section. Because mast cells are connective tissue elements, it was perhaps natural that their response to adrenal corticosteroids should be investigated since these hormones inhibit inflammatory reactions so effectively. Qualitative changes pertaining to variables such as release or clumping of granules, differences in staining properties, or cellular numbers in tissues are very difficult to evaluate reliably. The technical difficulties and the likelihood of introducing artifacts of technique are so numerous that it is not surprising that so many conflicting reports have been published. Mast cells are apparently damaged very easily,

for example by such a procedure as that involved in preparing a tissue-spread preparation, and even by pinching the skin.[14] The close apposition of mast cells to connective tissue fibers renders them particularly subject to mechanical tearing forces during teasing of the spreads, shrinkage of fibers in coagulating fixatives or dehydrating solutions, or spreading of paraffin ribbons prior to mounting on a slide. Even in air-dried smears of peritoneal fluid, I have observed under direct microscopical observation that mast cells shrink appreciably upon flooding the slide with 48/80.

Enumeration of mast cells in sectioned material is dependent on tissue density and composition at the start of manipulations. It is to be anticipated that tissues from adrenalectomized animals will differ from those of intact controls (or from those of donors on adrenocorticoid hormone therapy) in many important respects such as hydration, quantity of ground substance, resilience and size of collagen and reticular fibers. These differences may well account for many of the observations made. It was recognized by Baker[88] that an apparent increase in omental mast cell numbers following cortisone treatment was due to loss of fat and consequent shrinkage of the tissues. This problem is particularly obvious in lymphoid organs that are known to involute strikingly in response to glucocorticoid stimulation.[89,90] Kelsall and Crabb[91] recognized this difficulty and attempted to circumvent it by counting mast cells in entire cross sections of thymus; this is only a partial correction, however, since involution (or shrinkage) must be a three-dimensional process and, in maintaining a constant section thickness, they failed to compensate fully for all spatial changes involved.

Against this brief and incomplete background of technical difficulties, it is easy to understand why reports of vacuolization, degranulation, or granule clumping following corticosteroid treatment[92-101] have not always been confirmed.[18,102-108] Cavallero and Braccini[109] felt the changes they observed in tumor mast cells were due to a pharmacological effect of cortisone. In peritoneal fluid, where more rigorous quantitation can be effected, mast cell concentrations are decreased after cortisone administration,[80] but this may be only an apparent change.[108] Their decreased numbers following adrenalectomy[9] can be evaluated only against sham-operated and replacement-therapy controls.[108] When absolute numbers are determined, the adrenal gland has not been shown to affect the mast cells of peritoneal fluid. Cytological parameters were affected similarly in operated and in sham-operated rats.[108]

Studies on hypophysectomized rats have shown that characteristic cytological changes can be elicited in mast cells of peritoneal fluid by endocrine factors. The changes attending hypophysectomy are not very rapid. The normal picture is restored to hypophysectomized rats by replacement therapy with whole pituitary implants or with a growth hormone preparation.[108] Other pituitary trophic factors have not been tested in this

system, but reports that adrenocorticotrophin (ACTH) affects mast cells of skin and mesenteries in adrenalectomized animals[105] suggest that this should be done. As already discussed above, physicochemical factors would favor a spherical shape (maximal volume to surface area ratio with minimal tensional forces at the surface) in cells freely suspended in a liquid medium. This situation actually prevails in the peritoneal fluid of intact, young, adult rats. Departure from the spherical shape observed for mast cells in hypophysectomized animals is therefore of particular interest. A more detailed discussion of these factors has been published.[108]

The advantages of using peritoneal fluid as a site for quantitative studies of various cellular types,[110] particularly mast cells, should be emphasized. Peritoneal fluid can be sampled by standard hematological techniques for determining cellular concentrations. This parameter, when coupled with measurements of total fluid volume present, allows the calculation of absolute cellular numbers.[18] Fluid volume can be estimated by direct measurement[40] or by weighing filter paper before and after sponging the cavity.[111] In addition, the physicochemical characteristics of the system (cells freely suspended in a low viscosity fluid) present special advantages in evaluating morphologic changes over the more conventional tissue sites (semigelatinous matrix with solid components).

One must guard against artifacts of technique but, in our hands, these have been less numerous when dealing with peritoneal fluid than with solid tissue. By most criteria, mast cells of peritoneal fluid are identical with their tissue counterparts. Wherever discrepancies have arisen on this score, it has been difficult to decide whether these were ascribable to the mast cells themselves or to their immediate environment.

Conclusions

Factual information pertaining to the mast cell is accumulating rapidly, but where it leads us is not clear. The function of these cells is still unresolved, and the long list of 25 hypotheses already advanced by 1938[5] has grown longer. Mast cells have become available in pure populations. This, coupled with the impressive development of microchemical analytical procedures, will undoubtedly yield much new information. In emphasizing difficulties of technique, I have hoped to use the critical approach necessary in reconciling and evaluating much of the conflicting information available. The present confusion, replete with a multiplicity of opinions and counteropinions, can be a positive force if it stimulates a more careful approach to work in this field.

Even before the function of mast cells is elucidated, these cells can serve in biological assays. This has been done for the quantitation of 48/80[63] and is suggested here for assaying activity of some alkaloid derivatives. Further investigation of the mechanisms involved in these procedures will probably broaden our understanding in the fields of cell

division, fat metabolism, anaphylaxis and inflammation, and perhaps collagen diseases.

I have restricted this discussion in such a way as to exclude any consideration of the basophil leukocyte. This should not be interpreted as an explicit contention that basophil leukocytes bear no relation to mast cells, a point I feel cannot be resolved at the present time.

Acknowledgments

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SECTION OF MATHEMATICS AND ENGINEERING

ELECTRIC ANALOGY STUDIES OF THE TRANSIENT BEHAVIOR OF HEAT EXCHANGERS*

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Introduction

Strictly speaking, all devices used for heating or cooling can be called "heat exchangers"; they require the exchange of heat between a heat source or sink and the object to be heated or cooled. In common engineering language, however, the term heat exchanger is reserved for apparatus serving the exchange of heat between two or more fluids, eliminating the cases of heat exchange between a solid and a fluid (as in a regenerator), or between two solids (pure conduction). Therefore we shall deal only with heat exchangers in this more narrow sense. Heat exchangers of this type find wide application in a number of industries, and the theory, as stated by P. Scholander[1] may have significance also in biological studies. For simple conditions, particularly if all design parameters are constant and if only steady state is considered, computational aids are hardly needed. The basic equations are to be found in textbooks on heat transfer, and reference is made here only to the publications by McAdams[2] and Schack,[3] the latter comprising graphs from which the solution of heat exchanger equations can be readily found. For some important cases of nonconstant thermal properties Colburn[4] has derived expressions that still permit relatively simple calculation.

Solution of equations becomes much more complicated if more than two fluids exchange heat in one apparatus. Problems of this kind led to the first attempts to solve the temperature distribution in a heat exchanger by means of electric analogy (Paschkis and Heisler[5]). The system used there will be briefly described below under *Circuit for Steady State Condition of Heat Exchangers*. Suffice it to say here that in this method of computation the physical length of the heat exchanger is replaced in the electric computing experiment by time. This method was later adopted by S. I. Juhasz and F. C. Hooper with hydraulic analogues.[6,7] The use of time in place of distance makes it impossible to use this circuit for the study of transient phenomena in heat exchangers. In the past such phenomena were of only limited interest to the designers of heat exchanger equipment, but for people who are concerned with automatic control of processes involving heat exchangers and with thermal stresses in the latter, the transient behavior of heat exchangers is im-

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portant. In order to handle this kind of problem, a new circuit was developed as described below under *Circuit for Transient Conditions of the Heat Exchanger.*

This circuit has been used repeatedly and, out of its several applications, considerations regarding the limitation of accuracy have arisen. The usefulness of the circuit can be extended by certain techniques in lumping, which are also applicable if the solution of the problem is to be carried out by finite difference methods. These considerations of accuracy and lumping are described below under *Lumping and Errors.* Finally, at the end of the paper under *Comparison of the Two Methods,* the two methods are contrasted.

Circuit for Steady State Condition of Heat Exchangers

Equations. The behavior of a heat exchanger can be described by EQUATION 1 for the hotter fluid, EQUATION 2 for the cold fluid (plus sign for parallel flow, minus sign for counterflow exchanger), and EQUATION 3 for the heat flow in the tube wall, neglecting axial flow; the notations are as listed at the end of the paper and, for better visualization, are also introduced in FIGURES 1 and 2.

Hot Side

$$E_h \frac{dt_h}{dx} + P_h h_h (t_h - t_{wh}) = 0 \tag{1}$$

Cold Side

$$E_c \frac{dt_c}{dx} \pm P_c h_c (t_c - t_{wc}) = 0 \tag{2}$$

Wall

$$k \frac{d^2 t_w}{dy^2} = 0 \tag{3}$$

Circuits. EQUATIONS 1 to 3 can be solved by measuring voltage and current in a circuit shown in FIGURE 3 for parallel flow, and in FIGURE 4 for counterflow exchangers. The tabulation below shows a set of analogies between the thermal conditions and the electric conditions, as well as a listing of equivalent units for thermal and electric units.

Thermal units	*Electric units*
Temperature (F.)	Voltage (v)
Rate of heat flow (Btu/hr.)	Current (microamp)
Thermal resistance per unit length (F. × hr. × ft./Btu)	Resistance (megohm)
Water value (Btu/hr., °F.)	Capacitance (microfarad)
Length (in axial direction) (ft.)	Time (seconds)

Obviously, if either the heat transfer coefficients or the flow rates of one or both fluids should change with temperature or position, computations become more difficult. Yet these kinds of problems can be quite readily handled by means of the electric computing circuits. If the specific heat is a function of temperature, then variable capacitances are used. If the boundary conductance is a function of temperature or

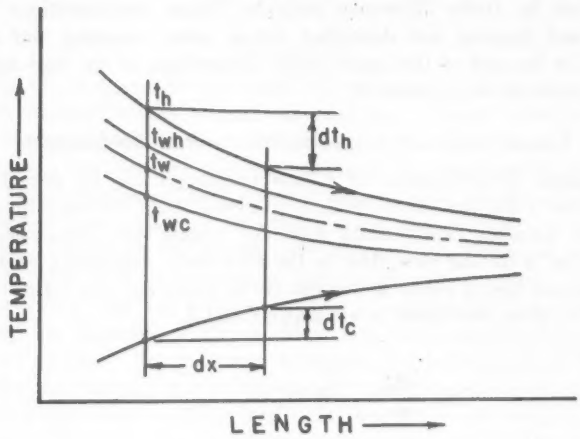

FIGURE 1. Steady-state temperature distribution in a parallel-flow heat exchanger.

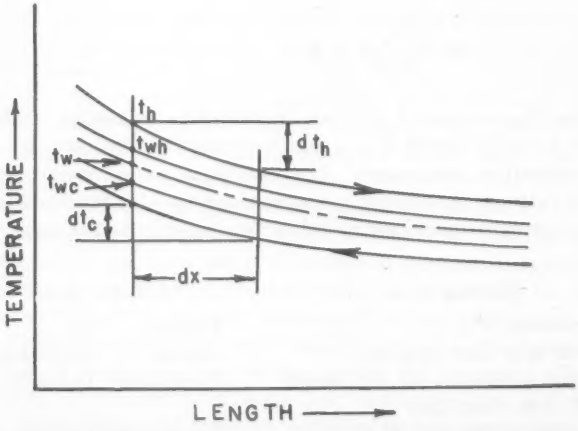

FIGURE 2. Steady-state temperature distribution in a counterflow heat exchanger.

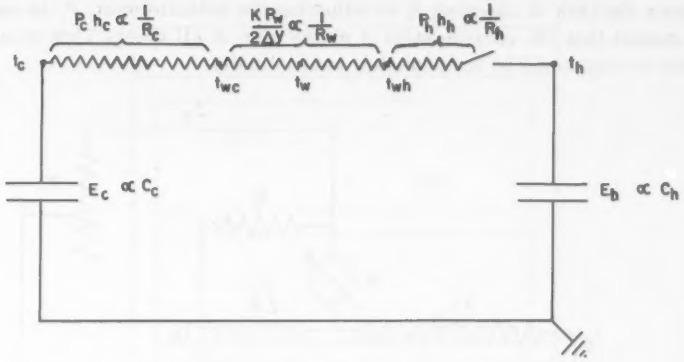

FIGURE 3. Electric analogy for steady-state parallel-flow heat exchanger.

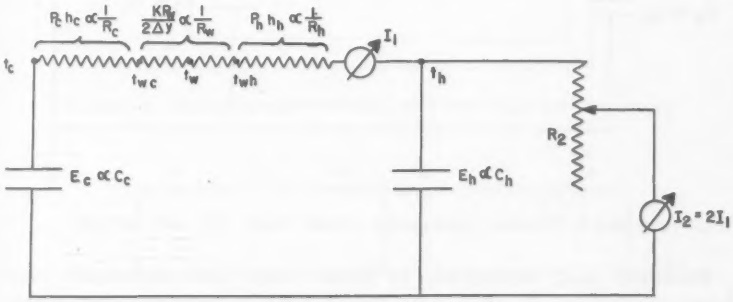

FIGURE 4. Electric analogy for steady-state counterflow heat exchanger.

position or if the conductivity of the tube wall changes sufficiently with temperature to make it necessary to consider such change, the resistance is broken down into the three components of which it is thought to consist: one resistance corresponds to the resistance to heat transfer from the hot fluid to the tube wall, the next represents the resistance of the wall itself, and the third the resistance from the wall to the cold fluid. One or more of these resistors can be made to vary during the computing experiment according to the appropriate voltage.

For the case of the counterflow exchanger the resistance R_2 is variable and adjusted at all times in such a manner that the current I_2 is twice the amount of the current I_1.

Operation of the circuit in such a manner that I_2 always has the correct value would be difficult unless the comparison of I_2 and I_1 is simplified. A null method has been devised, shown in FIGURE 5, which re-

duces the task of changing I_2 to adjusting the potentiometer, P, in such a manner that the microammeter A shows zero at all times. This adjustment to zero could be accomplished automatically.

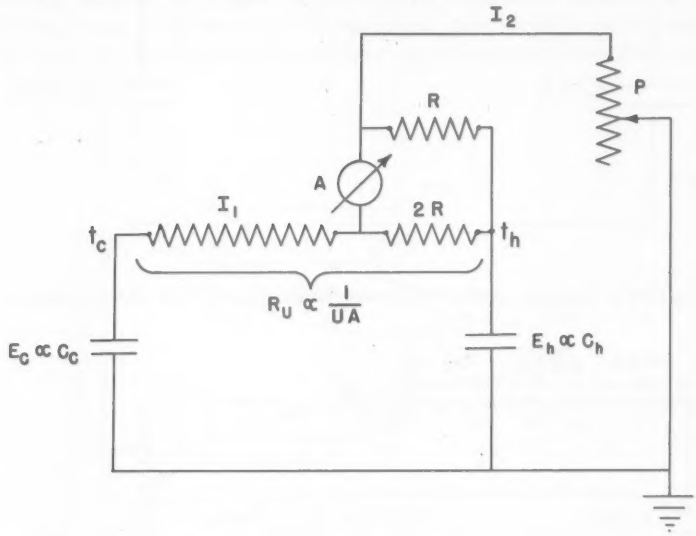

FIGURE 5. Electric analogy for steady state with null method.

Multifluid heat exchangers. In recent years heat exchangers have found application, in which more than two fluids exchange heat simultaneously. Reference is made to FIGURES 6 and 7, representing a three-fluid heat exchanger. Such exchangers are used, for example, in tonnage oxygen plants and, in that case, the three passages have gases according to the notation. If, as customarily will be the case, the channels contain an "extended surface," a thermal short circuit is provided across all the passages. It would be difficult to introduce this kind of arrangement in a conventional computation, but it has been solved successfully by means of a circuit shown in FIGURE 8. The individual resistances represent aggregates of several heat transfer resistances, as explained in detail in the publication by Paschkis and Heisler.[5]

In order to use this circuit for the solution of a problem, the temperatures of all three fluids must be known at one end of the exchanger. If the initial temperatures are not known for the same end of the exchanger, a trial-and-error method can be used by assuming end temperatures in some channels so that the computation starts with all temperatures at one end given. The computation is then carried out, determining the temperature at the other end of the exchanger. If the temperature thus

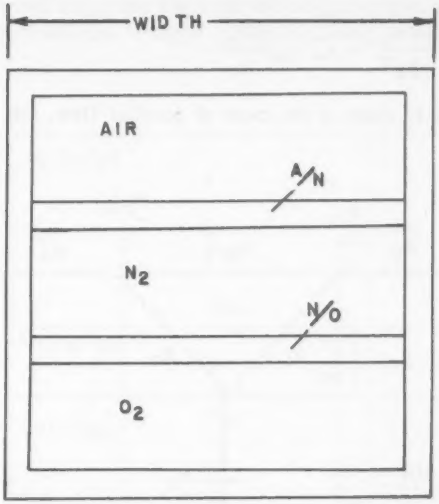

FIGURE 6. Schematic representation of a three fluid heat exchanger.

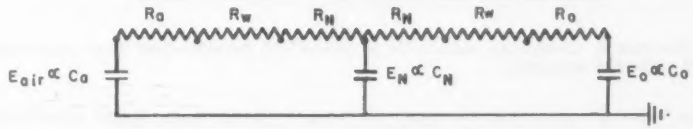

FIGURE 7. Steady-state parallel-flow analogous circuit for a three fluid heat exchanger.

determined does not coincide with the data, a new assumption for the entrance temperature is made and the computation is repeated. In view of the short actual computation time this procedure is still practicable.

Circuit for Transient Conditions of the Heat Exchanger

Equations. Disregarding heat conduction in the axial direction of the wall, the transient conditions of a parallel or counterflow heat exchanger can be represented by EQUATIONS 4 to 6:

Hot Side

$$A_h \rho_h c_{ph} \frac{\delta t_h}{\delta r} + E_h \frac{\delta t_h}{\delta x} + h_h P_h (t_h - t_{wh}) = 0 \qquad (4)$$

Cold Side

$$A_c \rho_c c_{pc} \frac{\delta t_c}{\delta r} \pm E_c \frac{\delta t_c}{\delta x} + h_c P_c (t_c - t_{wc}) = 0 \qquad (5)$$

Wall

$$k_w \frac{\delta^2 t_w}{\delta y^2} = \rho_w c_{pw} \frac{\delta t_w}{\delta \tau} \tag{6}$$

The plus sign is used in the case of parallel flow, the minus sign for counterflow.

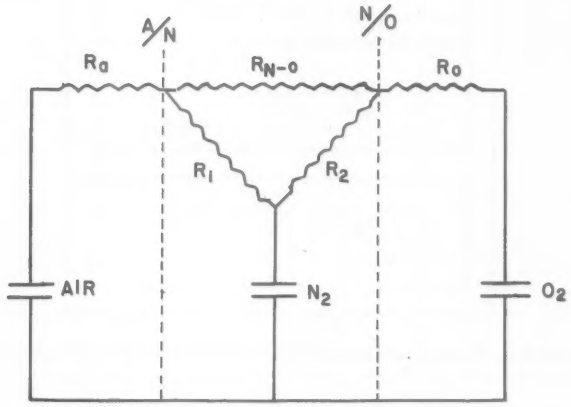

FIGURE 8. Computing circuit for a three-fluid parallel-flow heat exchanger with extended surfaces.

In order to represent the heat exchanger electrically, these equations are transformed into equations for finite difference in length but with the time variable in differential form. The length of the exchanger is divided into n sections or lumps (that is, $\Delta x = L/n$). Then the equations for the counterflow exchanger can be rewritten as follows, referring for nomenclature to FIGURE 9:

Hot Side

$$A_h \rho_h c_{ph} \frac{dt_h}{d\tau} + E_h \left(\frac{t_{h_2} - t_{h_1}}{\Delta x} \right) + h_h P_h (t_h - t_{wh}) = 0 \tag{4a}$$

Cold Side

$$A_c \rho_c c_{pc} \frac{dt_c}{d\tau} - E_c \left(\frac{t_{c2} - t_{c1}}{\Delta x} \right) + h_c P_c (t_c - t_{wc}) = 0 \tag{5a}$$

Wall

$$A_w \rho_w c_{pw} \frac{dt_w}{d\tau} + \frac{k_w A_w}{\Delta y} \left(\frac{t_w - t_{wc}}{\Delta y} \right) + \frac{k_w A_w}{\Delta y} \left(\frac{t_w - t_{wh}}{\Delta y} \right) = 0 \tag{6a}$$

In writing these equations it is assumed that the number of sections is sufficiently large to permit working with average temperatures for the

hot side of the wall (t_{wh}) and the cold side (t_{wc}) respectively; t_w is the average wall temperature, thought of as occurring in mid-thickness of the wall.

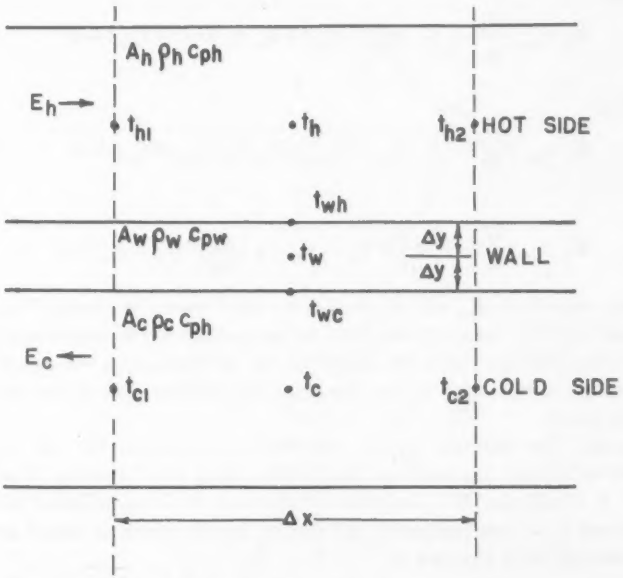

FIGURE 9. Element of a counterflow heat exchanger.

In practice the transient response of a counterflow heat exchanger is determined more readily by numerical or analogue methods if the exit fluid temperature of each section is used in place of the average temperature. On this basis the finite difference expressions for EQUATIONS 4 to 6 are written as:

Hot Side

$$A_h \rho_h c_{ph} \frac{dt_h}{dr} + E_h \left(\frac{t_{h2} - t_{h1}}{\Delta x} \right) + h_h R_h (t_{h2} - t_{wh}) = 0 \qquad (4b)$$

Cold Side

$$A_c \rho_c c_{pc} \frac{dt_c}{dr} - E_c \left(\frac{t_{c2} - t_{c1}}{\Delta x} \right) + h_c P_c (t_{c1} - t_{wc}) = 0 \qquad (5b)$$

Wall

$$A_w \rho_w c_{pw} \frac{dt_w}{dr} + \frac{k_w A_w}{\Delta y} \left(\frac{t_w - t_{wc}}{\Delta y} \right) + \frac{k_w A_w}{\Delta y} \left(\frac{t_w - t_{wh}}{\Delta y} \right) = 0 \qquad (6b)$$

Associating the elemental length Δx and thickness Δy with the respective cross sectional area perpendicular to the heat flow, EQUATIONS 4 to 6 become:

Hot Side

$$M_h\, c_{ph} \frac{dt_h}{d\tau} + E_h\,(t_{h2} - t_{h1}) + h_h\, A_h\,(t_{h2} - t_{wh}) = 0 \qquad (4c)$$

Cold Side

$$M_c\, c_{pc} \frac{dt_c}{d\tau} - E_c\,(t_{c2} - t_{c1}) + h_c\, A_c\,(t_{c1} - t_{wc}) = 0 \qquad (5c)$$

Wall

$$M_w\, c_{pw} \frac{dt_w}{d\tau} + \frac{k_w A_w}{\Delta y}(t_w - t_{wc}) + \frac{k_w A_w}{\Delta y}(t_w - t_{wh}) = 0 \qquad (6c)$$

In these equations M_c and M_h designate the "sectional weight" of the cold and hot fluid respectively. This is the product of the cross-sectional area of the passage times the length of the section times the density of the fluid. In determining M_w for the wall, the surface area of the section is to be used.

Circuits. The electric circuit, as shown in FIGURE 10, can be described by similar EQUATIONS (4d to 6d), using the following abbreviations: V = voltage; R = resistance (megohm); C = capacitance (microfarad); and τ_e = time (seconds). Subscripts are the same as listed above, in connection with FIGURE 9:

Hot Side

$$C_h \frac{dV_h}{d\tau_e} + \frac{V_{h2} - V_{h1}}{R_h} + \frac{V_{h2} - V_{wh}}{R_{hw}} = 0 \qquad (4d)$$

Cold Side

$$C_c \frac{dV_c}{d\tau_e} - \frac{V_{c2} - V_{c1}}{R_c} + \frac{V_{c1} - V_{wc}}{R_{cw}} = 0 \qquad (5d)$$

Wall

$$C_w \frac{dV_w}{d\tau_e} + \frac{V_w - V_{wc}}{R_w} + \frac{V_w - V_{wh}}{R_w} = 0 \qquad (6d)$$

The following tabulation shows corresponding units in the thermal and the electric system.

Thermal units	*Electric units*
Temperature (°F.)	Voltage (v)
Rate of heat flow (Btu/hr.)	Current (microamp)
1/Mass flow rate × specific heat (hr. °F./Btu)	Resistance (megohm)
also 1/Boundary conductance × surface area	
Weight × specific heat (Btu/°F.)	Capacitance (microfarad)

The two sets of equations (4c to 6c versus 4d to 6d) are analogous and equal if corresponding coefficients are equated. Because of the significance of these equalities for the error studies, a detailed discussion is found below under *Lumping and Errors*.

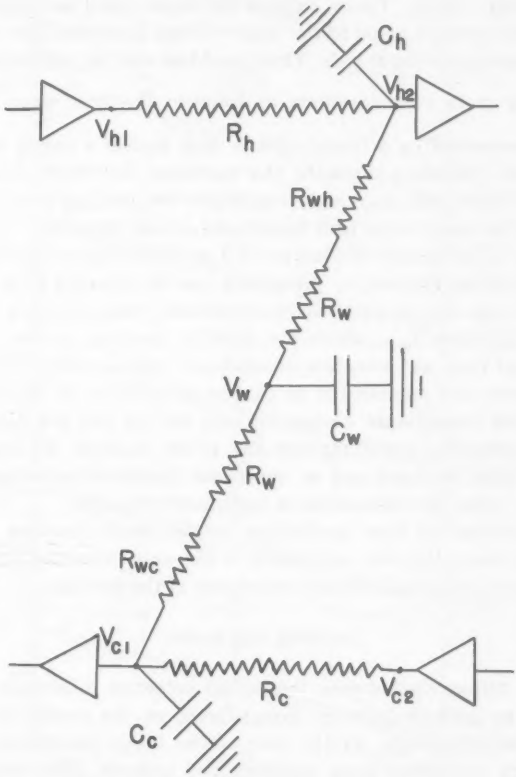

FIGURE 10. Circuit elements representing one section of a counterflow heat exchanger.

The Z-shaped lumping shown in FIGURE 10, resulting from EQUATIONS 4c to 6c, introduces a further approximation. In EQUATIONS 4a and 5a the rate of heat transfer from the hot side to the cold side in each section is determined by the average temperature of each section (t_h). This would require, in FIGURE 10, that the resistance R_{wh} be fed from a voltage equal to the arithmetic mean $(V_{1h} + V_{2h})/2$. Such feeding would require rather complex electric components. Provided there are sufficient lumps, the difference between V_{1h} and V_{2h} is small, and a small error

is incurred by feeding R_{wh} from V_{2h} instead of from $(V_{1h} + V_{2h})/2$. The error incurred by replacement of average voltages by end voltages in each section relates to accuracy and is discussed in the next section.

Representation of the finite difference equations requires placement of unity-gain cathode followers between consecutive flow resistors in the computing circuit. These cathode followers yield an output voltage (for the next section) equal to the input voltage (received from a section) without draining the input side. This procedure can be understood readily from steady state considerations as follows: the first order term $E\frac{dt}{dx}$ may be represented by a linear voltage drop across a single resistance. The cathode followers separate the sectional resistors, fulfilling the dictates of the equations, which require that the heat gain or loss across an element be equal to the heat transferred across the wall.

FIGURE 11 is the circuit diagram of a seven-section computing circuit for a counterflow exchanger. Transients may be induced by a change in mass flow rate and/or entrance temperatures; thus, FIGURE 11 shows switches S_{w1} and S_{w2}, which are used to simulate a step change in mass rate of flow and boundary conductance respectively. Step changes in temperature are represented by change of position of switch S_w. If a continuous temperature change occurs, say on the hot side input, a function generator would replace the power sources B_1 and B_2. No separate figure is shown and no equations discussed for a parallel flow exchanger, since the information is sufficiently similar.

Representation of heat conduction in the axial direction is accomplished by connecting the wall nodes in the axial direction by a resistor proportional to the axial thermal resistance of the section.

Lumping and Errors

(1) The difference between the actual potential difference and those found in the network becomes insignificant as the number of nodes increases and, therewith, as the size of the lumps decreases. However, since each additional lump requires two cathode followers, a large number of nodes becomes expensive.

Three methods are available to improve results for any given number of cathode followers (which determine the number of lumps). One is an extrapolation procedure; another is a method of using uneven lumps; the third is based on the concept that each lump is a separate heat exchanger.

(2) The extrapolation procedure has been described by Salvadori and Baron,[8] and reference is made to their text. For transients, the extrapolation would necessarily be carried out separately for each time increment that is to be measured. This greatly increases the number of extrapolation procedures and makes this method somewhat cumbersome for points inside the exchanger. It should be understood that the statements and

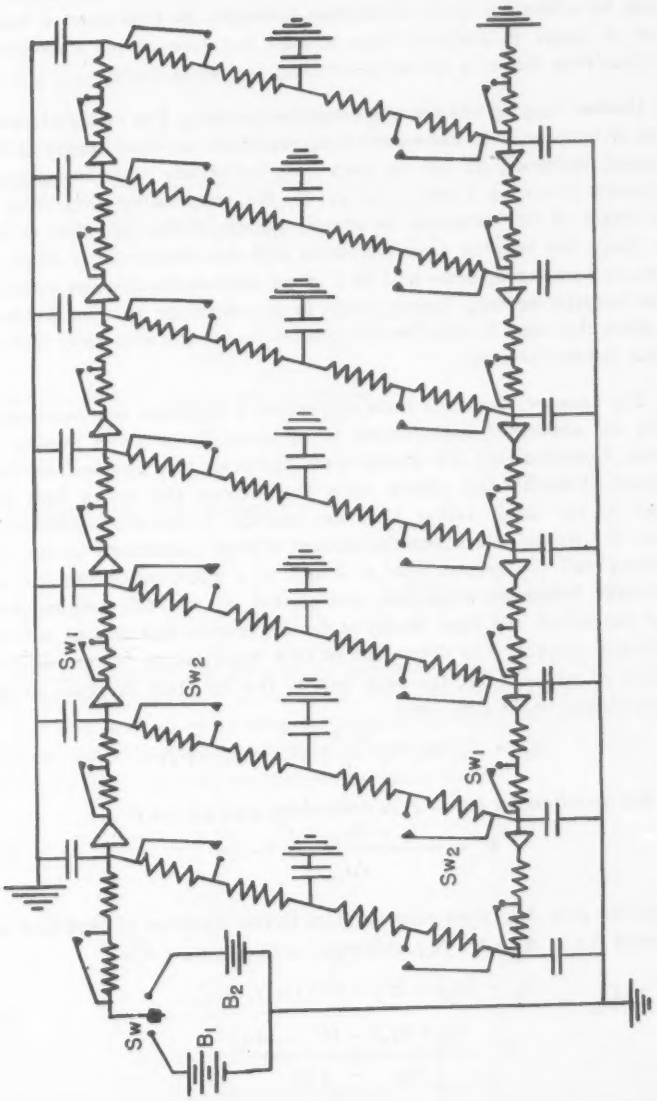

FIGURE 11. Complete circuit for counterflow heat exchanger.

conclusions in this section are made in terms of the electric analogy; however, these statements apply equally when one attempts the transient solution by classical finite difference methods. In this case a small number of lumps is desirable from a work and time-saving viewpoint, rather than from that of a saving in terms of equipment cost.

(3) Uneven lumping can improve conditions greatly. The straightforward method of lumping is to make each lump represent an equal length of the exchanger. However, as can be seen from the steady state temperature distribution (FIGURE 1 and FIGURE 2), the temperature drop along a given length of the exchanger is usually greater at one end than at the other. Since the lumping error increases with the steepness of slope of the temperature curve, there will be a larger error in the section with the greater temperature drop. Hence, there is an advantage in using unequal lump sizes that tend to equalize the temperature drops over each section and thus reduce the error.

(4) The concept that each node represents a separate heat exchanger permits an additional improvement in accuracy for a given number of sections. Consider first the steady state operation of a counterflow heat exchanger. Schack[3] and others show that across the entire heat exchanger a log mean rather than an average temperature difference between the fluids determines the amount of heat transferred across the separating wall. Therefore, what is sought is a modification factor that, in the finite difference equations, will correct, in part, the lumping error so that the initial and final steady state temperature distribution will be analytically correct. The derivation of this modification factor follows. Referring to FIGURE 12, the heat lost by the hot fluid is equal to the heat transferred to the cold fluid.

$$q_h = E_h \, \Delta t_x = \frac{U A_e}{F} (t_x - \Delta t_x - t'_x) \tag{7}$$

where the modification factor F is defined by EQUATION 8:

$$F = \frac{t_x - \Delta t_x - t'_x}{\Delta t_m} \tag{8}$$

The surface area A_e, taken perpendicular to the direction of heat flow of an element Δx is $A_e = P_w \ \Delta_x$; with equal sections $A_e = \frac{PL}{n}$

$$\Delta t_m = \frac{(t_x - \Delta t_x) - (t'_x - \Delta t'_x) - (t_x - t'_x)}{\ln \dfrac{(t_x - \Delta t_x) - (t'_x - \Delta t'_x)}{(t_x - t'_x)}} \tag{9}$$

The temperature ratio defining F can be related to the properties of the system by substituting the solutions for the exit temperatures. As given

by Schack[3] the hot side-exit temperature in the present notation:

$$(t_x - \Delta t_x) = t_x - (t_x - t'_x + \Delta t'_x)\frac{E_c}{E_h}(1 - f) \tag{10}$$

and the cold side-exit temperature

$$t'_x = t_x - (t_x - t'_x + \Delta t'_x) f \tag{11}$$

$$\text{where } f = \frac{1 - E_h/E_c}{1 - \frac{E_h}{E_c}\exp\left[-\frac{UA_s}{E_h}\left(1 - \frac{E_h}{E_c}\right)\right]} \tag{12}$$

Rearranging EQUATION 7:

$$F = \frac{UA_s}{E_h}\left[\frac{(t_x - \Delta t_x) - t'_x}{\Delta t_x}\right] \tag{13}$$

substitution of 10 and 11 into 13 yields:

$$F = \frac{UA_s}{E_h}\left[\frac{E_h}{E_c}\,\frac{f}{1-f} - 1\right] \tag{14}$$

and substitution of 12 gives:

$$F = \frac{UA_s}{E_h}\left[\frac{1 - E_h/E_c}{1 - \frac{E_h}{E_c}\exp\left[-\frac{UA_s}{E_h}\left(1 - \frac{E_h}{E_c}\right)\right]} - 1\right] \tag{15}$$

A similar procedure can be used to derive a correction factor for parallel flow, the need for which is somewhat less than for counterflow.

The use of F in the analogous network is straightforward in that the resistances (thermal or electric) corresponding to the hot-side film, cold-side film, and wall resistance are multiplied by F.

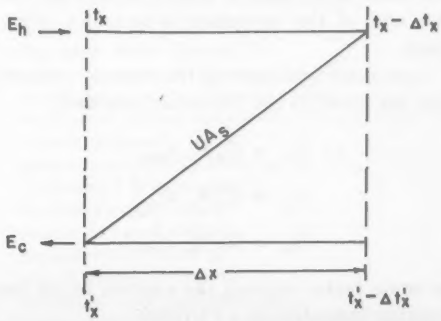

FIGURE 12. Notations used in derivation of modification factor.

The use of the correction factor leads to exact answers for the steady state and improves results for the transient conditions between two steady states.

Heat transfer coefficients that are functions of position are directly accounted for in this concept. The assumption is made that each element has a constant UA_s, but UA_s may differ from section to section.

The circuit values in the analogous network, as mentioned earlier, are determined by relating the coefficients in EQUATIONS 4c to 6c to the coefficients of EQUATIONS 4d to 6d.

$$R_h = \frac{R_\epsilon}{E_h} \qquad (16)$$

$$R_c = \frac{R_\epsilon}{E_c} \qquad (17)$$

$$R_w = \frac{F\,\Delta y\,R_\epsilon}{k_w\,A_w} \qquad (18)$$

$$R_{hw} = \frac{F\,R_\epsilon}{h_h A_h} \qquad (19)$$

$$R_{cw} = \frac{F\,R_\epsilon}{h_c A_c} \qquad (20)$$

In these equations, R_ϵ is the scale factor relating the electric to the thermal resistances; it has the dimension (megohm $\times$ Btu)/(hr. $\times$ °F.). The factor F is the modification factor, as per EQUATION 15; if the number of sections is sufficiently large, F becomes nearly 1, indicating the decrease in lumping error.

The corrected values give the exact temperatures for the steady state, if the hot-exit temperature of each section is higher than the cold-exit temperature. If, because of the small number of lumps available, some of the sections are too large for this condition (hot exit higher than cold exit) to be satisfied, then it is impossible to obtain the exact exit temperatures. EQUATION 15 reflects the impossibility by demanding a negative value for F (that is, a negative resistance!). In this case the best that can be done is to make F equal to zero for the least critical sections at that end of the exchanger where the rate of temperature change is smallest.

The electric capacitors representing the thermal capacity of the fluids and wall material are found by the following relations:

$$C_h = C_\epsilon M_h c_{ph} \qquad (21)$$

$$C_c = C_\epsilon M_c c_{pc} \qquad (22)$$

$$C_w = C_\epsilon M_s c_{pw} \qquad (23)$$

where C_ϵ is the scale factor relating the electric to the thermal capacity; C_ϵ has the dimension (microfarads $\times F$)/(Btu).

For the convenience of reading on the analogue computer, the electric

computation experiment need not be carried out in the same length of time considered for the exchanger; a time scale is introduced into EQUATIONS 4d, 5d, and 6d. This time scale, noted as TR, is dimensionless and is defined as the ratio of electric time to heat time. It is related to the scale factor R_ϵ and C_ϵ by the following equation:

$$R_\epsilon \; C_\epsilon \; \times \; \frac{1}{3600} \; = \; \mathrm{TR} \qquad (24)$$

Comparison of the Two Methods

Obviously the first method, in which exchanger length is represented by time is simpler for steady state problems; there is no problem of spatial lumping, and high accuracy can be obtained, even for the complex cases of more than two channels and temperature-dependent properties.

The second method, including spatial lumping, is more versatile; it permits handling of both steady-state and transient problems with or without axial heat flow in the tube wall. It also allows easier visualization because positions on the tube can be identified in the circuit. However, in order to obtain a steady state accuracy comparable to that of the first method a large number of nodes would be required.

Acknowledgments

H. G. Landau of this laboratory was consulted on several of the mathematical aspects and suggested the use of the modification factors.

The circuits for transient behavior of heat exchangers were developed under grants by the Allison Division of the General Motors Corporation, Indianapolis, Ind., and the Harrison Radiator Division of the same firm, Lockport, N. Y.

Nomenclature

A	Area perpendicular to heat flow (sq. ft.)
C	electrical capacitance (microfarads)
C_p	specific heat of fluid (Btu/lb. °F.)
$E = Wc_p$	water value (Btu/hr.°F.)
f	abbreviation as per (EQUATION 12)
F	modification factor (EQUATION 15)
h	coefficient of heat transfer (Btu/hr., sq. ft., °F.)
I	current (amperes)
k	thermal conductivity (Btu/hr., ft., °F.)
L	total length of exchanger (ft.)
M	sectional weight (lbs.)
n	number of equal sections
P	perimeter of channel (ft.)
q	rate of heat flow (Btu/hr.)
R	electrical resistance (megohms)
t	temperature (°F.)
U	over-all coefficient of heat transfer

V	voltage (volts)
W	mass flow rate (lb./hr.)
x	distance parallel to direction of flow (ft.)
y	distance perpendicular to direction of flow (ft.)
Δy	half thickness of wall
ρ	density (lb./cu. ft.)
τ	time – actual heat time (hr.)
τ_e	time in the electric circuit (sec.)

Subscripts: h, hot side; c, cold side; w, wall; wc, wall cold side; wh, wall hot side; e, electric; and s, surface of element.

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*SECTION OF PSYCHOLOGY**

RESPONSE PATTERNS**

By R. C. Davis

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Although one can easily obtain wide agreement that psychology is the study of behavior, specifications of the meaning of behavior vary considerably. Frequently a piece of behavior is distinguished by an effect it has upon the organism's, or better, on the animal's environment. Consequently we have experiments in which rats press a bar, and in which humans press a key, move a lever, and speak words. In these events we distinguish one response from another; we score "success" or "failures" according to what occurs in the environment. Although such a starting point is a proper one, on brief reflection it may seem a surprising one. Starting thus, we group together those actions of the animal that have the same effect (this, of course, is the principle of response equivalence). It seems similar to a classification of diseases according to the amount of fever they produce; on the basis, that is, of result rather than process.

It is recognized, to be sure, that various processes may achieve the same result. In fact, to some individuals, this consideration seems to justify such a classification. The end is everything, the means are nothing. However, it is no great surprise to find that the point is controversial. A well-known minority proposes that there is greater lawfulness about means than about ends, that we should do better to note the details, at least the particular limb movements, of an animal's actions.

This proposal makes a truly sporting controversy, because the facts known are so scarce that either or neither side could be right. There is a challenge to grasp the club and hand ax and join the fray, but a gentler call to take up the quest for more of our scientific substance.

The special question before us can be put quite briefly: "Can a detailed description of responses and their antecedents throw any light on an animal's action?" This question does not pose a choice of physiological information as opposed to some other sort, about either effects or causes. The question of which facts are more valuable seems hardly worth considering: one would evidently become tangled in theories of marginal utility, and in our kind of horse race it is the performance of the field that really matters.

A "detailed description" could mean simply a listing of observable

*At a meeting held by the Division of Mycology on May 24, 1957, Michael L. Furcolow presented a paper entitled "Recent Studies on the Epidemiology of Histoplasmosis." This article will appear as an Annal shortly to be published by The New York Academy of Sciences.

**This paper, illustrated with lantern slides, was presented at a meeting of the Section on May 20, 1957.

limb and head movements. Such a description, however, is still only a partial one, and modern instrumentation allows us to go much further. Any response one can observe with the unaided eye seems to have a wide penumbra of other components that can be brought to light by recording instruments. The records show muscular activity not only in the moving and supporting musculature, but on a smaller scale in many remote muscles; they also show concomitant changes in the other organ systems of the body. For example, consider what occurs when a subject presses a key in response to a signal, the classic simple reaction experiment, with a prior set of instructions and a warning signal. Muscular activity begins to increase shortly after the warning signal and, with the signal to respond, develops very rapidly both in the acting muscles and in the remote parts, according to a certain pattern. When the pattern presents a large enough difference between flexor and extensor action, the key is closed, and muscular activity soon begins a decline in a regular decelerated curve. Meanwhile a certain pattern of changes is occurring in other sectors: in respiration and in the autonomically controlled processes.[1] For example, sweat gland action increases, breathing becomes faster and deeper, the heart slows down, the blood vessels in the hand constrict, and pulse pressure goes up. Probably all of these changes are initiated by the warning signal. To a great extent the warning signal, if sufficiently strong, will produce such responses even if no action is required of the subject. If the subject is required to press a key, the total pattern of these responses is stronger and, qualitatively, somewhat different.

In this total complex the fact that the key was pressed appears as a direct consequence—an important one, of course—of just one small feature of the pattern: the local muscular contraction. One should ask, at this point, whether the rest of the information, as I have given it, will contribute anything to our understanding of the key press. It might be argued that this set of facts is interesting for its own sake because it shows something of the total energy expenditure that goes with the activity. In addition, however, will this kind of data help in solving the problem of whether the key press, or more generally any overt behavior, will occur; when it will occur, how forceful it will be, and how it may be modified in accordance with the experimenter's wishes? There *are* some promising signs of relations between what one may call the pattern and the overt response. First, a stronger, larger pattern of the right sort may be indicative of a quicker, stronger response. Second, two patterns leading to different consequences may overlap, and the amount of identity in the two could be the basis for facilitation, response generalization, transfer, conditionability, and other response interactions. Third, in the organism there are patterns that follow stimulation, but are not accompanied by any action, and patterns that remain after an action has taken place. These hidden patterns may incline the individual to execute one action rather

than another, and either help or hinder the action that is the resultant. These cases may be systematized as, first, the one system case, involving just one response pattern; second, the case of two or more simultaneously activated systems; and, third, the case of one or more preactivated systems. It would be possible to give data to illustrate each of these relationships, but there is a question basic to all that should be answered. In each of the three "theories" there is a presumption of a considerable variety of different excitation patterns. Therefore, I propose to examine this question.

Half a century or more ago there would have been very little hesitation in answering it. Though the problem was formulated in quite another way, the answer, I think, would have been that different patterns occur when there are different feelings or emotions. Wilhelm Wundt had the expression of feeling and emotions, as he called it, well systematized with very few facts. His logic was quite simple: by introspection he would identify the fundamental aspects of feeling, and then corresponding states in the body must be found by the principle of parallelism. On his first point modern evidence makes it clear that Wundt was wrong: somatic response patterns are not restricted to such states as he called feelings and emotions, but are continually present. Further, his identification of affective states by introspection was much too facile. Other introspectors could and did say, "He is wrong; I observed it otherwise," and about the only reply an outsider can make is, "What are you fellows talking about, anyway?"

Actually, I doubt if there has ever been a thoroughgoing introspective attack on the number and character of the emotions. A list of them used to be considered essential in textbooks (and, alas, is still thought valuable, if the number is not greater than three[2]). However, one gathers that such lists were actually constructed backward. The mental states alleged were inferred from situations and from actions by some rather loose methods, probably influenced in great measure by popular tradition and the availability of certain names in the European languages. Even if exact introspection were possible (a condition contrary to fact, as the grammarians would say) it would not enable us to solve our problem. Self-observation could hardly be expected to reveal all the variety of the response patterns, nor can behavior nowadays be taken as a simple manifestation of a state of consciousness. We may as well, therefore, come to the point directly on response patterns on their relation to stimuli, and on overt responses.

Another overhasty answer to the question of pattern differences has had a long vogue, partly, I think, because of the forcefulness of W. B. Cannon. Cannon[3] had no doubts of emotions as mental states, nor of their variety, but he concluded on anatomical and physiological evidence that body states, in the viscera, at least, could not vary correspondingly. In these

there would be only a state of vegetative functioning, or an emergency state in one degree or another, the one dominated by parasympathetic neural activity, the other by sympathetic. This extreme simplicity of action is suggested first by the manifold cross-connections within the sympathetic division and by the fact that more than one sympathetically innervated organ can be seen to respond at the same time. Then, in Cannon's own experiments, cats were found to have, so far as could be observed, the same visceral state when exposed to four or five kinds of severe stimulation. In the light of present knowledge, the argument is short of being conclusive. The parasympathetic nervous system would now have to be recognized as capable of producing excitatory states also, and various combinations of the two autonomic branches seem to be the rule.[4] We should not feel very safe in functional inferences from anatomy, and there is doubt, at least, that the sympathetic nervous system acts uniformly in all parts. Cannon's own observations of response variables were too limited in number and exactitude. For a clarification of the general question, the skeletal muscles, the sweat gland reactions, and a number of bodily states in addition to those that Cannon observed would be equally interesting. As a result we are face to face once again with the need for more facts on the pattern of responses.

The kind of work required is technically difficult in data collecting and data treatment, and expensive in time and money, but there is now an increasing number of laboratories in which it is being carried on. Apparatus engineering, data handling, and statistics all need technical improvements, but they are advanced sufficiently to make experiment possible where it would have been inconceivable a few years ago.

Different patterns in skeletal muscle action and tension are not difficult to find and relate to stimuli and responses. The problems become more difficult when other response variables are included. A first line of inquiry, including some of the autonomic variables, concerned individual peculiarities in response, called response or symptom specificities. Malmo and his co-workers[5] showed that patients with psychosomatic symptoms have their favored ways of reacting to questions about their lives and problems. The patient with psychosomatic heart trouble actually responded with circulatory system changes, and the patient with headaches responded with muscle tension in the head region. Lacey, Bateman, and Van Lehn[6] were able to show that normal persons likewise have their idiosyncracies. By an ingenious statistical method they demonstrated that an individual exposed to several kinds of stimuli will have his greatest response in one measured variable more often than can be accounted for by chance. Certain results in our laboratory provide further confirmation.[7]

Correlating responses with individuals in this way gives assurance that the pattern of somatic response is not fixed and invariant for the species. The stereotyping found in individuals might, of course, derive

from the inheritance of differing anatomical structures. There is, on the other hand, the interesting possibility that patterns of reaction are subject to modification by some kind of learning, and that a person develops his own stereotype. This learning would be more than the attachment of a new stimulus to an old response; it would be a modification of the structure of the response itself with the promise of eventual intentional shaping of the responses. I do not believe we have, as yet, an experimental investigation of how this might take place in the total response pattern.

Much of interest remains to be discovered concerning these individual peculiarities, but the general questions about the relation of response patterns to overt responses cannot be answered with this kind of evidence. We must proceed to inquire about the possible variety of response patterns in the same individual on different occasions. Does a person's excitation pattern change from time to time, and are these changes referable to conditions that an experimenter can manipulate?

In the Lacey, Bateman, and Van Lehn experiment each person's response to a stimulus was expressed as a deviation from the average response to that kind of stimulus. This treatment is clearly a proper one for the study of individual differences. The very first step in the treatment is one which eliminates differential effects of stimuli that otherwise would confuse the issue. If we wish to study the effects of different stimuli on the same person, we should reverse the procedure and temporarily eliminate from consideration, the differences between individuals. The experiments on individual differences show that some of the variance of responses is attributable to individuals. Whether there is also some variance attributable to stimuli and to interaction remains an open question.

One experiment carried out by Ax[8] a few years ago gave promising results in showing a difference between two autonomic reaction patterns. The experimenter placed the subject in some rather diabolically contrived situations while he made his recordings. A general description of these and of the subject's behavior led to an identification of the patterns as "anger" and "fear." There is, I fear, some danger in using those familiar words, at least until their reference is made fairly definite. Nevertheless, it seems that two different excitation patterns were provoked by some features of the situations used in this experiment.

In our laboratory we have been carrying on certain experiments that are concerned similarly with somatic patterns. In setting up the experiments we have tried to be quite noncommittal about the number and variety of patterns to be expected. We are not, in other words, looking for the "correlates" of such patterns as "fear," "repulsion," and "sex emotion." The independent variables in the experiments are therefore explicitly the stimulus and surrounding conditions, as I suspect is always fundamentally true in this sort of experiment.

We began our studies with some very simple stimuli and with instruc-

tions to the subject that he was to do nothing.[9] While we varied the stimulus in certain systematic ways, we recorded about a dozen somatic response variables. Our method for detecting pattern change among these variables was to plot each one as a function of the stimulus variable, when that could be quantified. If these functions are similar, we assume that a pattern of response changes in intensity, but not in structure. This is approximately what happened when we varied the intensity of a brief auditory stimulus. As we approached the pain threshold the size of the responses increased faster than did our decibel input, but the responses all kept pace with one another, maintaining, as it were, the same formation. This pattern we called by an arbitrary name: the *N* pattern. We found one way in which this *pattern* could be modified. If we repeated stimuli at one-minute intervals, some components of the pattern adapted faster than others. Generally, the components with shortest latency are most affected by adaptation. The principal respiratory response, which has a very long latency, actually increased with repetition. The result at the end of the series was necessarily a rather different response pattern.

Simple visual stimuli, according to a later experiment,[10] also produce an *N* reaction. However, with cutaneous stimuli there is a difference. These, especially pressure and warmth, seem to produce a vasodilation effect in the periphery, where the *N* pattern calls for constriction. Our strategy of pattern detection was necessarily different in this study, because our stimuli varied qualitatively rather than quantitatively. We applied the Friedman test of agreement in ranking, a method that has been developed further in later studies. For all types of cutaneous stimuli we found that the autonomic pattern remains about the same, but that the skeletal muscle response varies in a different manner from stimulus to stimulus. For all there is an increase in tension, but not an increase proportional to the autonomic components. The typical reaction to cutaneous stimuli we designate as the *C* pattern.

We proceeded then to some more complicated situations.[7, 11] In one experiment the stimuli were projected pictures for the subject to look at, and in the other he was required to perform the mild task of paced key-pressing.

In the picture experiment our Friedman test shows, for male subjects, a significant agreement in the way the several autonomic variables are affected by the stimuli. This means that we have something like the first, general factor of a factor analysis. The pattern, which may be called the typical picture or *P* pattern, is one of great sweat gland activity, peripheral vasoconstriction, and cardiac deceleration. This pattern is produced in its purest form in male subjects presented with a picture of a nude female. There is good reason to think that other pictures have somewhat different effects, that there are, in other words, factors other than the first in our matrix. Certain response variables have good reliability correlations, but they are only slightly related to the first order pattern.

The picture of a starving man produces, not only less response, but a different sort of response with increase rather than decrease of pressure pulse, and a reversed effect on the breathing measures. These are all results for male subjects, and those for females differ from them. In our measures taken together the female response is less, even when we disregard the pictures of nudes and, so far as our statistical tests go, we cannot say, for all measures taken together, that females distinguish one picture from another. Nevertheless, I am convinced that they really do. For if we look at certain response measures, heart rate, for example, we find that females actually respond more than males, and respond differentially. This fact brings to light an important relation: an interaction between response measure, kind of subject, and the stimuli. This fact also shows that response patterns are no simple matter.

The important result of the experiment on paced tapping is, for the present purpose, the occurrence of a new dominant pattern, apparently associated with mild exercise. We have called this result the *E*-1 pattern because it is almost but not quite what one gets on a larger scale in vigorous activity according to standard physiological experiments. The one big difference is in the behavior of the peripheral vasculature, which constricted in our experiment instead of dilating as it does in treadmill running.

There are in our experiments four major response patterns that we may compare. To be precise, description of a pattern requires that a relation between the components be stated, and to compare patterns would be to compare relationships. However, I do not quite know how to do so at this point, but we can simplify. In TABLE 1 the patterns are described simply by using signs to show whether the function concerned is more or less

TABLE 1

COMPARISON OF RESPONSE PATTERNS

(Double signs indicate diphasic effects)

Pattern	*E - 1*	*N*	*P*	*C*
Palmar-sweating	+	+	+	+
EMG	+	+	+	+
Pulse rate	+	+ -	-	-
Volume pulse	-	-	-	-
Pressure pulse		- +	-	+
Finger volume	-	-	-	+
Chin volume		+	-	+
Respiration rate	+	-	-	+ -
Respiration amplitude	+	+	-	- +

active than in the resting condition before each stimulus. Where there are diphasic effects double signs are used. In the first two rows of the table, and in the fourth row, we see features common to all patterns. It looks as though any stimulus would produce an increase in sweat gland and muscular activity. I do not recall having seen or heard of an opposite effect. The case for volume pulse is more doubtful. More vigorous activity might reverse its response, for one thing, and for another it seems that for cutaneous stimuli the decrease means maximum vascular dilation, rather than constriction. In the other rows, columns differ a good deal from one another. Apparently the general effect of the pictures, aside from the first two variables, is to depress the activities measured. By contrast, the tapping task increases most of them. The *N* and *C* patterns are intermediate and mixed, differing from each other in the heart rate and possibly in the breathing reactions.

It is convenient to speak of patterns as though they were a denumerable set of entities, distinct from one another. I have no reason to think this is true. All the variables in the pattern are continuous, and it seems most likely that the patterns would likewise merge into one another in a series of gradations if conditions were varied appropriately. The several variables, the ones we have recorded and others, could be represented in a hyper space, with some dimensions assigned to stimulus variables and others to the response variables. Then, as we moved along a stimulus axis, it is doubtful that the accompanying response variables would behave in discontinuous, saltatory fashion.

We should expect, as a further complication, that the structure of this hyper space would change as a result of learning. Certainly, the responses become attached to new stimuli and detached from old ones, and they may change with respect to each other. Many of the problems about response patterns can be put as questions about the distribution of points in this complex space. There is, for example, the matter of a general activation state, to use E. Duffy's idea.[12] Suppose we regard the sleep state as the point of origin in all our dimensions. Then the question becomes, "Will a movement along any stimulus dimension cause us to move on all response dimensions, in the same way on one as on another?" Perhaps there is a common solid in some dimensions around the origin. Our muscle tension and galvanic skin response results given above suggest this might be a true picture, although we do not know the size of the solid.

This imaginary space is helpful, I believe, in forming problems, but naturally an experimenter could manage only a small part of it at a time. He would need to make a judicious slice somewhere and apply the microscope to the section.

We are certainly in need of the experimenter's information. Our knowledge of somatic patterns is very meager indeed. I think we do know enough of them, however, to say that earlier guesses of one, two, or

three modes of variation are a long way short of the facts. Tangled as the relations are, there is an assuring hope for us in their very complexity. The peripheral patterns may provide us with a way of exposing to direct light the wealth of intermediate processes that we can suspect in a person, but cannot otherwise see.

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SECTION OF ANTHROPOLOGY

AN ANALYSIS OF THE POLITICAL ORGANIZATION OF THE MOSSI PEOPLE*

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The African Sudanic states were the subject of a discussion presented before The New York Academy of Sciences in 1949. At that time Joseph Greenberg discussed the rise of these states and evaluated the theories assigning the predominant part in their formation to Caucasian influences. However, the Negro genesis of the Mossi Sudanic state never has been seriously questioned.[1] In this paper I propose to analyze the political system of the Mossi in the light of such questions as: the power structure of the African state; the conquest theory of state formation; and the presence of feudalism or feudal-type states in the Sudan.

The one and one-half million Mossi represent the largest population aggregate in what is now known as French West Africa. They inhabit an arid 125,000 square miles of sub-Saharan area drained by the Black, Red, and White Voltas and their tributaries. The Mossi are described physically as "true" Negroes as distinguished from the other Sudanic types. Their language belongs to the Mossi-Grunshi groups of the Niger-Congo family of African languages. In many respects their culture is similar to those of such Voltaic groups as the Tallensi, Dagomba, and Mamprussi peoples of the Northern Gold Coast (Ghana). The people in this Voltaic region are mainly agriculturalists who grow several varieties of millet and sorghum. They rear some cattle, sheep, and goats, but do little fishing or hunting. Their kinship systems are based on patrilineal segmented lineages. Throughout this entire region we find the institution of earth priests who are ritual owners of the land, but political organization is highly segmented and acephalous, with the exception of the centrally organized systems of the Dagomba, Mossi, and Mamprussi.

The *Tarikh es Soudan*, a chronicle written by Es Sadi, who lived in Timbuktu during the early seventeenth century, records that the Mossi first appeared on the historical scene about 1328. Between the years 1328 and 1477 the Mossi of the northern kingdom of Yatenga attacked and conquered Timbuktu, sacked Macina, and raided the towns of Bagana and Walata held by the Songhoi. In turn they were defeated by Sonni-Ali, the famous Songhoi ruler, who even invaded their territory, capturing slaves and booty. Askia El Hadj Mohamed, ruler of the Songhoi, raised a holy war

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against the Mossi in 1497 and defeated them, but he failed to make them embrace Islam. The Mossi and Songhoi fought several other wars until Songhoi power was broken by the Moroccans, who captured Timbuktu in 1590.[2]

The Mossi first came to the attention of the Europeans during the travels of Heinrich Barth, J. Dupuis, and Edward Bowdich, but the first European, G. A. Krause, did not enter Mossi country until 1888. Krause was followed six months later by Louis G. Binger, but the Mossi became suspicious and closed their territory to other Europeans. About 1894, one Ferguson, a British mulatto, safely arrived at Ouagadougou, the Mossi capital, and signed a treaty with the supreme Mossi ruler placing the area under British protection. The French arrived in the country in September 1896 and, refusing to honor the British treaty, conquered the Mossi and secured control of the country.

The Mossi traditional history goes back to about the eleventh century and is part of a tradition relating the origin of the Dagomba, Mamprussi, and Mossi peoples. The Mossi claim descent from Nyennega, the daughter of a Dagomba ruler, who fled from her father's home to marry a hunter by the name of Riallé. This man had left his father's country and was living in exile as a result of a quarrel over the chieftainship. The product of the union between Riallé and Nyennega was a son called Wedraogo in honor of the stallion that carried his mother to the north. At the age of fifteen the boy Wedraogo went back to the Dagomba to visit his grandfather and was given 4 horses and 50 cows. According to tradition, when Wedraogo left the Dagomba country to return north many Dagomba horsemen followed him, because their country was overcrowded. Wedraogo and his followers rode northward and conquered his father's people, the Boussansi, and decided to live in a place called Tankurn (Tengdodogo=old country). Here Wedraogo married a Boussansi woman called Pouiriketa, who bore him three sons. Diaba Lompo, the eldest, was sent out to rule Fada N'Gourma; another, called Rawa, was sent to rule a place called Zondema (now part of Yatenga); the youngest, Zoungrana, remained with his father at Tengkodogo.

Tradition states that the Boussansi were only one of a group of small but warlike peoples who inhabited this region. The others were the Ninisis, the Kibissi, and the Gourounsi. These groups were constantly at war with one another and they made frequent alliances with stronger peoples to fight their enemies. A Ninisi chief visited Tengkodogo and, seeing the strength of the Dagomba, sought an alliance with Wedraogo. The Ninisi chief sent Wedraogo a woman called Pouitenga as a wife for Zoungrana, Wedraogo's son. A few years later the Ninisi asked Wedraogo to come and rule their country, but he died at a place called Sagabtenga before he could establish complete control of their territory. Zoungrana succeeded his father as head of the Mossi (a new tribe resulting from the union of the Dagomba and the local inhabitants) but, wishing to remain at Tengkodogo, he sent his son Oubri to complete the conquest of the

country. The Ninisi were happy to have Oubri as their sovereign since he was related to them through his mother. Oubri made war on the local inhabitants and subjugated or drove away those Ninisi, Kibissi, and Gourounsi who did not accept his rule.

Naba Oubri is regarded as the founder of the Ouagadougou dynasty, the most important of the dynasties issuing from the original Dagomba conquerer, Wedraogo. The Mossi rulers who succeeded him spent most of their lives subjugating the rest of the country; the sites of their graves are markers along the road of Mossi conquests. It was only after the reign of Naba Coibga, the fourteenth ruler, that the Mossi rulers were able to confine their activities to the capital at Ouagadougou. By the reign of Naba Warga, the twentieth ruler, ritual prevented the leader from leaving his capital. The country had become so centralized at this time that it was feared that civil strife would erupt if the sovereign left his capital.

The consolidation of the Mossi state seemed to have been a difficult task, and at various periods in its history some decentralization took place. During the reign of Komdimie, the fifth ruler, two of his relatives broke away from his control and formed principalities. The northern kingdom of Yatenga was created by a man called Yadiga, and the state of Riziam was founded by Kouda. Despite opposition and constant attacks by Komdimie the new states remained independent and Yatenga absorbed Zondema, the fief of Wedraogo's son. At this same period Komdimie allegedly gave the provinces of Boussouma, Yako, Conquisitenga, and La to his sons, who called themselves Dima, meaning "Submit only to God." Another province called Kayao broke away from Komdimie's control, and its sovereign took the title of Dima when the supreme ruler was unable to conquer him. Most of the Dimas established courts similar to that of the supreme ruler and performed similar functions, but they still recognized the supremacy of the Ouagadougou dynasty. Besides these small independent states ruled by the Dimas, there were the important independent entities of Tengkodogo, Fada N'Gourma, and Yatenga. The first two claimed independence from Ouagadougou because they were founded by the sons of Wedraogo, while Yatenga claimed independence by the right of revolt and by the subsequent absorption of Zondema. The rulers of these states were forbidden to see each other, or to see the Ouagadougou ruler on pain of death inflicted by their ancestors.

The traditions that describe how the Mossi rulers subjugated and controlled the country differ little from region to region. As the rulers conquered new areas they sent their sons, in company with the sons of their ministers, to command the new districts. When the country was finally conquered the new rulers sent their sons to rule districts taken away from the heirs of former rulers.

The people of Nobéré, the district in which this study was carried out, say that their first sovereign, Biligo, was heir to Oubri, but that he did not wish to rule over the Mossi. Biligo left Ouagadougou on a hunting expedi-

tion and, when he arrived at the sparsely settled Ninisi village of Nobéré, he decided to stay and make it his home. The Ninisi ran away when they saw Biligo coming, but he persuaded them to return and live in peace under his command. The Tengsoba, or earth priest, of the village refused to return, and Biligo went into the bush and captured him so that the sacrifices to the earth shrines could continue as before. Biligo married a local woman and, when she bore him sons, he sent them to rule those villages not inhabited by the earth priests. These sons and their families formed the noble classes in the district.

About one hundred years ago a district chief called Peka supported the loser in a fight for the supreme chieftainship and was killed when the victorious pretender was installed as chief in Ouagadougou. The victor sent his son, Ngado, to rule over Nobéré and, although the relatives of Peka were scattered, the village chiefs were allowed to retain their posts. These chiefs did lose their noble status, however, because Ngado's sons who went to live in the villages formed the new nobility. The chiefs and their families were submerged into the commoner class. The history of Nobéré is typical of that of most of the Mossi districts and, today, one finds the remnants of many former ruling lineages scattered around the country.

The supreme ruler of the Mossi was called the Moro-naba. He possessed the *nam* which is the symbol of power, and because of this power he had the right of life and death over all his subjects. He also had a religious role in the country because it was his duty to initiate the yearly rites dedicated to the ancestors so that the country would have good crops, and so that the people would be happy and multiply. The Moro-naba was aided in his rule over the Mossi by five ministers who were responsible for the five provinces grouping 333 villages that made up the territory of the state. These ministers were responsible for the proper administration of the state, and they nominally elected the new Moro-naba.

As soon as the death of the ruler was announced, civil disorder broke out throughout the country and continued until the five ministers and the minister for war took fictive control of the country. When the funeral of the late chief was completed the chief minister, the Ouidi-naba, summoned the lesser ministers to a conference and told them that the country had no chief "because the fire had gone out" and that it now required one. He advised them to consider the qualifications of the late chief's son and of all the other pretenders, and to choose the most worthy individual. The lesser ministers refused to do this, saying: "You are our superior, and the right to make the choice rests on you alone."

The eldest son of the former ruler was usually elected Moro-naba but, as any member of the royal lineage up to the third degree was eligible, many persons tried to bribe the elector. When the Ouidi-naba made his choice he told the minister of war: "My mind is not at ease because I fear that those whose hopes have not been fulfilled will resort to violence."

The war minister replied, "That will be against tradition and justice to the dead ruler. Do not fear; I am here with my arms to see that your decision is respected" (this was not always an empty gesture, for one man, Wobogo, forced a Ouidi-naba to elect him).[3] When the lesser ministers were notified as to the choice of the new sovereign they said: "You have expressed our will and the will of all the people." The elector then secretly called the new ruler and, taking off the man's goat's skin (the symbol of those seeking the chieftainship) told him: "I am giving you the house of your father and of your grandfather." Then the ministers and the chief musician escorted the new ruler before his subjects, and the Ouidi-naba shouted: "Here is he who has the power of life and death over you." The following day all the members of the ruling lineage and sublineages swore not to betray their new ruler. The minister of war then swore, in the name of all the people, to protect the new ruler with his life if the need ever arose. Next followed ceremonies during which a descendant of the original Ninisi inhabitants made sacrifices, in the name of the new sovereign, to his ancient ancestors.

In analyzing the power of the Moro-naba we must take into consideration the concept of *nam* which is described as "power" or "the force of God." It is this force of God that enables one man to control another, and gives the sovereign the power of life and death over his subjects. Moreover, once a man had "eaten the *nam*," the Mossi constitution had no provisions for deposing him. The Mossi generally claim that only one of their thirty-three rulers was ever deposed, and that he was the ruler who ran away from the French. However, there is another tradition that states that one sovereign, Karfo, was forced to commit suicide by his Ouidi-naba.

According to the Mossi constitution, the Moro-naba had complete control over the lands of the state, and was the only one empowered to distribute these lands as fiefs. Any attack on the lands of the state was an attack on the Moro-naba and thus a cause for war. The Moro-naba alone had the right to make war, and in this he was helped by his vassals, the district chiefs, and their armies. He was court of final appeal for crimes that carried the death penalty and, theoretically, only he had the right to condemn a nobleman to death.

Every year the district chiefs under the command of their ministers went to the capital to pay allegiance to the Moro-naba with gifts of cows, goats, sheep, cowries, and grain. The animals were presents from the stock of the district chiefs, but the cowries and grain were collected from the villagers of the districts. The people of the several wards in the capital furnished labor for the cultivation of the sovereign's fields and for the upkeep of his houses. The Moro-naba received substantial sums of money from taxes on the sale of slaves or other merchandise in the Ouagadougou market. He also received presents of slaves when his vassals raided the surrounding countries. If a district chief failed to fulfill all of these

obligations to the Moro-naba, his lands were raided, his women seized and, if he were caught, he was killed.

It is difficult to ascertain how the institution of provincial administrators arose among the Mossi. Some of the people still believe that the ministers were formerly slaves of the Moro-naba, but Delobsom claims that the first ministers were warrior companions of Naba Oubri, the founder of the Ouagadougou dynasty.[4] These men were not members of the royal lineage and thus were inferior in status to the members of the royal lineage and sublineages under their supervision. However, as emissaries of the Moro-naba, these ministers were entitled to respect by all the people. The ministers recommended successors to vacant district chieftainships; thus it was incumbent upon a district chief to be friendly with his minister if he wished succession to go to his eldest son.

The minister was the person who carried orders from the Moro-naba to the district chiefs, and he reported back to the Moro-naba on conditions in the district he visited. However, the minister could not interfere in the internal affairs of a district, nor could the district chief complain to the Moro-naba about a minister. Everyone was required to observe the strict hierarchy; the only sanction that the district chief exercised over his minister was to appeal to another minister in the hope that the Moro-naba would come to his aid.

The districts that comprised the provinces of the ministers were scattered around the country and were not equal in size or importance. One minister, the Baloum-naba, controlled the districts ruled over by those chiefs who were the sons of recent rulers. Another, the Kamsoro-naba, a eunuch, had control of the largest and wealthiest districts, but he, in contrast to the other ministers, was not succeeded by sons and, when he died, the Moro-naba inherited all of his property. The ministers frequently became very wealthy because they received gifts whenever their charges went to Ouagadougou to pay homage to the ruler. The people from the districts under their control provided labor for their fields and for the upkeep of their houses. The ministers also received bribes from persons seeking vacant district chieftainships.

When a district chief or Kombéré died, his heirs sent a horse or a sheep with leaves on its tail to notify the ruler of the death. The Moro-naba always questioned the envoys, and when he was satisfied that his man had not met with foul play, he sent the animal to his chief minister. The Moro-naba then sent his own envoys to the late chief's house with offerings for sacrifices to their common ancestors, and also instructed the envoys to collect the death offerings from the chief's relatives. After the funeral the eldest son of the dead chief and the other pretenders went to Ouagadougou bearing gifts to the district minister and the other important men surrounding the ruler. When the time came to name the new district chief, the Moro-naba called his ministers together and asked them: "Which one of the candidates is really capable of succeeding the late chief?"

The Ouidi-naba answered: "Naba, you may do as you desire." The ruler then said, "Under these conditions I will designate the eldest son of the late chief as the new Kombéré because his father has never forgotten me, or gave me any trouble when he was alive. It will not be good to reject his son." The ministers then called the heir and put the following question to him three times: "Before his death, did your father designate anyone to replace him as chief?" The heir replied three times: "No! My father did not designate anyone. You may name anyone you wish; even if he is a blind man or a leper." The heir was then given a bonnet, the symbol of the *nam*, and made to swear on drinking the Tibo, a ritual potion, that if he ever betrayed the ruler, may the Tibo kill him. Thereafter, all the members of the new chief's lineage swore to respect and obey him.

The power and function of the Kombéré were similar to those of the Moro-naba, but on a much reduced scale. As the person who had received the *nam* from the Moro-naba, he was supreme in his district, subject only to his obligation to the head ruler. These obligations included the customary gifts and aid in warfare. There is some evidence that many of the Kombérés were lax in paying this tribute and rendering homage to the Moro-naba and consequently were punished or killed. Naba Warga fought a five-year-long civil war against a rebellious vassal before the rebel was killed, and Naba Wobogo was engaged in a three-year civil war with one of his vassals when the French arrived in the country. It is not clear whether the Moro-naba was able to prevent or stop war between his vassals over strayed cattle or stolen women. The Mossi in the district of Nobéré said that the Moro-naba did not often know when neighboring villages were at war. These wars did not involve the loss of territories by the districts, since it was ritually forbidden for one district chief to take lands from another.

The district chiefs had ministers who helped them administer the districts. These ministers were descendants of the sons of the Moro-naba's ministers who accompanied the first district chief when he took up his fief. They had control of several villages and they channeled goods and services from the villages to the district heads. The hierarchy of command was also strictly observed on this level, and a village chief who had disputes with other village chiefs, or who had important cases to be judged, was required always to be accompanied by his minister when he visited the district chief. This official could put any man into stocks for serious crimes, but he could authorize the death only of commoners. It must be pointed out, however, that in many cases the district chief usurped the rights of the Moro-naba, and had a troublesome noble secretly strangled. The district chief, like the Moro-naba, could not be deposed; he was often forced to commit suicide or met death by foul play or in open civil war. The people in the district reacted to an unjust ruler by leaving his territories.

The village chiefs held their fiefs directly from the district chiefs, and

were enfeoffed through the principle of primogeniture on the advice of district chiefs' ministers. They paid homage, and owed fealty, to their district chiefs, although part of their tribute always was sent to the Moro-naba. In the event of war they followed their district chief when he went to serve the Moro-naba. The village chiefs received presents from the heads of the families in the village and settled any local court cases in their villages. They did not have the right, however, either to put men into stocks or to have them executed. They were aided in the administration of the villages by the heads of families.

Almost every Mossi village had sections in which the nobles lived. As stated above, these people were the relatives or descendants of the district heads, and they lived in a semiparasitic condition upon the commoners in the village. Most of them owned horses and, in addition to furnishing the cavalry for the district chief's contingent during war, they raided and attacked caravans passing through the country. The nobles were subject to the control of the village chiefs, and it was not unusual for them to be under the jurisdiction of chiefs who were members of the commoner class. In such cases the district chiefs had a great deal of difficulty resolving the problems between their vassals and their kinsmen. The nobles could steal the wives and daughters of the commoners, but if a commoner tried to steal the wife of a noble he and the woman were both killed. Whenever a noble threatened to disrupt the life of a district he was secretly strangled by the district chief's men.

The above is a brief account of the origin and structure of the Mossi political system. Most of the myths regarding the origin of this system seem to suggest that here is a classic conquest state, but one neither founded by Caucasians nor extraordinarily influenced by them. The Mossi state is homogeneous in race, language, and culture, a feature not found in most of the other Sudanic states. This fact is not interesting in itself until we note that the Mossi share many cultural traits with the Voltaic peoples. Marc believes that this cultural homogeneity is the result of the Mossi invasion of the area during which they imposed their languages and customs on the indigenous populations. Apparently not believing the traditions about the Dagomba origin of the Mossi, he explains the fact that such non-Mossi groups as the Nankana, Wala, and Dagari speak Mossi dialects as proof that the Mossi were politically more powerful in ancient times, but "the feudal regime disappeared little by little.... The Mossi language was kept; [it is] the only actual witness of the ancient power of the Mossi" (author's translation).[5] We know, however, that the Mossi were still encroaching on the territories of their neighbors when the French arrived in 1896.[6]

Rattray believes that the inhabitants of Mamprussi, Dagomba, and Mossi territories are the same now as they were "centuries before the ancestors of those who are now their rulers appeared on the scene."[7] He states that

these conquerors even adopted the language and culture of the indigenous populations, but introduced an "administrative machinery" that welded scattered local groups into loose confederacies without affecting in any great degree the inner life of these groups.[8] Rattray's hypothesis appears to explain quite adequately the reasons for the cultural homogeneity of the Mossi and the other Voltaic peoples. It also conforms to what we know from recent historical times of the organizing and conquering abilities of such figures as Samory and El Hadj Omar. These men carved out veritable empires in this area in remarkably short periods. However, we need not accept Rattray's hypothesis that foreigners were the ones who instituted "administrative machinery" among the Mossi. It is highly possible that the Mossi state might have been the result of internal Voltaic development when one Dagomba lineage established its hegemony over scattered local people in this area. There is some evidence here that the Mossi state had many of the features of the widespread Negro pattern of political structure so common to the other Sudanic states, and this fits in very well with what Professor Greenberg reports about the basic African nature of these states.[9]

It is clear that the Mossi state was a complex entity and many persons cannot describe it without calling it feudal.[10] However, the question of feudalism itself is a vexing one, and many scholars limit the use of this term to describe the political system of medieval France and possibly Japan. In fact, not many people would admit the presence of feudal states in Africa although some, such as S. F. Nadel, who worked among the Nupe, call the Sudanic states feudal and talk about fiefs and vassalage. It is now generally agreed that feudalism, despite its other aspects, is basically a form of government. Stephenson gives the classic features of a feudal state as: (1) fief-holding with vassalage; (2) homage and fealty; (3) mutual obligations between lords and vassals; (4) military service from the vassals; (5) inheritance of fiefs by heirs of vassals, usually by primogeniture; (6) subinfeudation; (7) attendance at court; and (8) wardship and marriage arrangements by which fiefs could be held in trust for minors and, in the case of female heirs, husbands who can render homage for them.[11]

An examination of the Mossi political system in terms of the first six characteristics of feudalism shows that this system conforms very well to the specifications for a feudal system. The Mossi did not have an elaborate court structure on the European model and, since only mature males could inherit fiefs, no arrangements for wardships and marriages were necessary. Furthermore, I do not think that these two traits are necessary for feudalism, and they can be considered as cultural content that need not be present in all social processes with similar functions.

Mossi-like societies present a special problem to political theorists because of the importance of kinship in their political systems. Coulborn[12] strongly insists that "A feudal society... is based on contract. It is not

based on kinship." He contrasts the Geselschaft feudal system with the tribal Gemeinschaft, and recognizes the possible transition from Gemeinschaft relationships to Geselschaft relationships in the evolution of culture. However, this admission places Coulborn on the horns of a dilemma as he does not accept feudalism as a step in the evolution of the state, but as a form of government that develops after the breakdown of a state and is used for state restoration. He states that "The place of feudalism in the rise of civilized societies is not yet complete, but we must pause to notice the profound importance of the substitution of something different for kinship relations."[13] We see from the Mossi data that they used kin bonds instead of warrior-band bonds as the basis for delimiting the persons chosen for government. The warrior-band members were rewarded with hereditary administrative positions. It must be emphasized, however, that despite kinship bonds, it was the loyalty of a man that determined whether his heirs received his fief.... These new vassals were required to renew pledges of allegiance to a ruler sometimes very remotely related. It must be borne in mind that kinship does play a part in political systems that range from feudalism to twentieth century Western democracy.

We cannot fail to observe that it was in regard to the society of the Chou period in China, one similar to that of the Mossi, that Coulborn was compelled to withhold judgment as to the nature of its political system. Shang society seemed to have had many of the tribal characteristics of the people found in the pre-Mossi Voltaic area. Chou had to face the problems of ancestor veneration associated with the tribal lands, and used some of the same techniques as the Mossi. Coulborn found many of these features "foreign to a tribal society" and finally decided to "leave open the decision as to whether Chou China developed a feudal system or a system of city states as its first enduring polity."[14]

In conclusion I shall say that the political system of the Mossi had definite feudal characteristics. These feudal characteristics did not come as a result of the breakdown of a pre-existing state, but served as techniques for building a state from small scattered groups. I should certainly like to keep open the whole question of feudalism, especially as it applies to the question of the origin of the pristine state, until more comparative data from Africa can shed additional light on this important subject.

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SECTION OF OCEANOGRAPHY AND METEOROLOGY

THE STORM SURGE PROBLEM IN NEW YORK CITY*

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Introduction

Ever since man living along the seacoasts first began to notice the periodic rise and fall of the water and the action of the wind upon it, he has been fascinated and at times terrified by storms that have brought water surging in along the coast well above the high-tide marks. It has since become a well-established fact that a wind blowing from the ocean will tend to raise the water level and that an offshore wind will tend to lower it.

This effect has been defined as the meteorological tide or the storm surge. Strictly speaking, any deviation from the normal predicted astronomical tide that is due to meteorological conditions is called the meteorological tide. Some writers (Miller[1] and Schalkwijk,[2] among others) claim that differences in atmospheric pressure must be regarded as playing some role in the ocean's maintenance of hydrostatic equilibrium with the atmosphere. Theoretically, at least, a hurricane that has a central barometric pressure one inch of mercury lower than the pressure outside of its periphery should have a central water level approximately one foot higher. The opposite view of G. Neumann, New York University, New York, N.Y., holds that in a storm at sea the water is pushed tangentially away from the center and that, accordingly, the atmospheric pressure effect is nullified and negligible. However, when a hurricane or deep extratropical storm moves over a semienclosed body of water such as Long Island Sound, N. Y., and crosses the coast, the effect of pressure is complicated both by the nature of the coastline and by the narrow channels, such as the Race and the East River, available for the mass transport of water. In a few cases, an additional surge has accompanied the passage of the hurricane center over the coast line (Redfield and Miller[3]).

The Problem

Hurricanes and extratropical storms along the seacoast cause more damage through water action than through wind action. Buildings can be constructed to withstand the force of the wind, but very few houses of ordinary construction can take a battering from the sea. Erosion from waves and currents causes considerable damage once the water level is raised to the point where it can reach installations inland. By far the

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greatest damage to property and automobiles during the hurricanes of 1938, 1944, and the three in 1954 was due to tidal flooding and the subsequent action of waves and currents. Many boats were battered to pieces at their moorings or broken up when they were swept inland.[4]

Although hurricanes are the most publicized and the most spectacular of storms, some disturbances of extratropical origin have brought surges to the New York coast that are as high as or higher than many of the hurricanes of recent history. Two storms are worthy of mention – that of November 25, 1950, and the combined wind and snow storm of November 7, 1953. TABLE 1 shows a comparison of water heights for stations in the New York surge forecast area for various hurricanes and storms since 1938. It may be seen readily that the two extratropical storms that occurred in November hold the record for the highest surges at many stations in this area.

Following the 1954 hurricane season, more funds were allocated to the Weather Bureau for research on the hurricane problem. One of the sections that was set up was the Storm Swell Section with specialists assigned to various key centers along the Atlantic and Gulf coasts. The main object of the research is to establish such empirical relationships as would be useful in forecasting tidal surges from established data or from forecasted data that would be available at the time the surge forecast is made.

The emphasis was placed on empirical methods at the outset since it was obviously necessary to come up as quickly as possible with some answers to the question: "How high will the surge be?"

Theory has not progressed very far in providing a solution and, although some theoretical work has been done, the field is still in a primitive stage. It is to be hoped that eventually the differential equations for the surge can be set up to yield solutions by means of modern machine methods, but the empirical approach presently offers the best hope. With the latter technique we are faced with the age-old cry of all empirical research men: we need more data.

In attacking the problem, it was necessary first to set down what we know about it. From theory and experience, we know that a wind blowing over water in the northern hemisphere will set up a current and bring about a mass transport of water to the right. After a period of minimum duration, steady winds will establish an equilibrium between wind and the level of the sea. If the same wind continues to blow, there will be no appreciable change in the meteorological tide. This is comparable to the minimum duration period used in the forecasting of waves by the Pearson-Neumann-James method.[5] In this method, the height of the significant waves remains the same for a given wind speed after the period of minimum duration has been exceeded.

The effect of the wind on the sea is affected greatly by the shape of the coast line and the depth of the water (Marmer[6]). The wind has its

TABLE 1

MAXIMUM SURGE LEVELS (M.S.L.) FOR MAJOR STORMS AND HURRICANES, 1938-1955

Station	*Sept. 21 1938*	*Sept. 15 1944*	*Nov. 25 1950*	*Nov. 7 1953*	*Aug. 31 1954*	*Sept. 11 1954*	*Oct. 15 1954*	*Aug. 12-13 1955*	*Oct. 14-16 1955*	*Mean high water*
Fort Hamilton, N. Y.	6.4	6.7	7.5	7.7	5.9	4.3		4.5	6.4	2.3
Perth Amboy, N. J.	6.6	7.4	9.5	8.8	5.8	4.8	5.5	5.3	7.7	2.6
Spuyten Duyvil, N. Y.	5.3	6.0	7.0	6.7	5.2	4.0	4.2	4.4	5.9	1.8
Lawrence Point, N. Y.			10.0	10.4	10.9	6.2	5.9	5.2	7.4	3.2
The Battery, N. Y.	6.4	6.4	7.4	7.6	5.6	3.8	4.5	4.2	5.9	2.1
Sandy Hook, N. J.	5.9	8.4		7.9	6.1	4.4	4.6	4.1	6.2	2.3
Willets Point, N. Y.	12.0			8.7	11.4	6.4	6.1	5.3	7.8	3.5
Montauk (Fort Pond Bay), N. Y.	15.8	7.9		5.4	7.9	3.6	3.1	2.1	3.9	1.0
New London, Conn.	9.8	6.1	7.2	5.9	8.7	3.0	4.0	2.5	4.2	1.2

greatest influence on the surface of the sea. This effect decreases rapidly from the surface downward.

In *Dynamical Oceanography* J. Proudman[7] gives the inclination of the surface of the water by the following formula:

$$\text{Sin } \alpha = \frac{F_s}{g \rho h}$$

where F_s has the dimensions of stress, g is the acceleration of gravity, ρ is the density of water, and h is the depth of the water. As may be seen readily from this formula, for similar wind stresses water will rise higher in a shallow body of water than in a deep one.

Hubert and Clark[8] list three potential surge-producing features in hurricanes:

"(1) A wind-driven mass of water is driven up on the coast. This is frequently referred to as set-up.

(2) Convergence of wind driven currents in the sea.

(3) Elevation of the sea surface under the low pressure of the hurricane – the 'inverted barometer' effect.

"These forces can act independently and if superimposed can produce additive effects."

The surge in the open deep water is probably quite small. When the hurricane or extratropical storm approaches the coast, however, the kinetic energy of the wave of translation of a long advancing wave is concentrated on a small portion of the coast line. Hubert and Clark[8] state that this is usually caused by (1) refraction of wave energy by the outline of the continental shelf and (2) funneling of water in bays and estuaries.

R. O. Reid and B. W. Wilson[9] have computed the set-up as a function of maximum wind, the length of the fetch and the slope of the continental shelf along the path of the advancing storm. However, the authors point out that the following simplifications were used: (1) the bottom was assumed to be a simple inclined plane; (2) the storm was assumed to be symmetrical and advancing normal to the coast; and (3) the distribution of wind stress along a line parallel to the storm's path is assumed to have a single simple shape.

Another phenomenon that may be important but that is difficult to estimate quantitatively is the convergence of the drift currents (Hubert and Clark[8]). Due to the distribution of wind and the frequent presence of shear lines across which the wind changes abruptly in a hurricane, the surface of the sea may be raised when a fast drift current converges with a slower drift along a line. This may not be significant when the water is deep and compensation takes place at lower levels but, in shallow water, this convergence may attain appreciable proportions.

Amplification by funneling results from the forcing of water into bays

and estuaries. This will depend to a large measure on the configuration of an enclosure such as Long Island Sound. It has not been possible as yet to point to a specific difference in meteorological tides between stations in Long Island Sound or New York Bay and state that this difference is due to funneling alone.

Refraction of waves by bottom topography where the wave front travels in a direction perpendicular to the bottom contours is mainly an ocean wave phenomenon and generally entails little water transport. However, at some particular station on the coast where the bottom contours are V-shaped with the point extending out into the water, a concentration of wave energy at the station due to the two advancing and converging wave fronts will pile up more water at the station than at points some distance away on either side.

Seiches, or surface oscillations, in enclosed or semienclosed basins are other phenomena that must be considered. If the winds can impress a seiche of the proper frequency on such a basin, significant amplification may result. In Buzzards Bay, for instance, the winds in Hurricane Carol pushed the water up to the northwestern end of the bay, the maximum height of 12 feet occurring at 1130. The water was released as the storm passed northward. As a result, the water acted as a uninodal standing wave and surged back on the southeastern end of the bay, attaining maximum heights at about 1300. Surge heights reached 15 feet on the southeastern end of the bay and occurred *after* the hurricane had passed. Incidentally these were the greatest heights ever recorded for the Cape Cod area. It is likely that the westerly winds also had some effect on the surge. Marmer[6] has computed that the natural frequency of Long Island Sound is approximately 11.8 hours. Since the periodic force of the ocean tide is about 12 hours, one can expect it to set up and maintain a stationary wave in Long Island Sound. The characteristic features of the tides and currents in the sound show that this is true. The so-called "resurgences" that occur in Long Island Sound after the passage of a storm have not been fully studied, but it is quite possible that these may be due to oscillations of a multinodal standing wave. Redfield and Miller[3] show that the maximum water levels developed in Hurricane Carol by the Long Island Sound resurgence factor decrease from 8 feet in Block Island Sound to 5 feet at Bridgeport and rise again to more than 8 feet at the entrance to the East River. This distribution is quite different from the normal high water tidal distribution that ranges from approximately 1 foot (M.S.L.) at Montauk Point to nearly 3.5 feet (M.S.L.) in the western end of the sound.

Theoretically, at least, the principle of resonance plays a part in adding to the surge height. Proudman[7] derived the expression

$$\xi = \bar{\xi}/(1 - \frac{C^2}{gh})$$

for the amplifying effect of coupling between a gravity wave and a traveling atmospheric wave, where ξ is the height of the water wave; $\bar{\xi}$ is the equilibrium water wave height; g is the acceleration of gravity; h is the depth of water; and C is the speed of the atmospheric wave.

Since $\sqrt{gh}$ is the speed of the long wave, the right side of the equation approaches infinity as the speed of the water wave approaches the velocity of the atmospheric wave C. In nature, due to the irregularity of the bottom contours, the very short time the low spends over a particular contour, and the fact that the equation is essentially applicable to a one-dimension infinitely long canal of uniform depth, we do not get pure resonance with these storms. However, some evidence is present to show that the resonance factor probably accounts for some portion of the surge height in some hurricanes.

Research

Since extratropical storms produce surges that range in a continuous spectrum from the lowest significant values to those encountered in hurricanes, the first investigations were made on such disturbances. As a first approximation, it was hoped that results from these investigations would also be applicable to the hurricane surge problem.

A listing was first made of those simple parameters that might contribute to the meteorological tide. These were: (1) the pressure gradient (favorable directions); (2) the wind velocity (favorable directions); (3) the phase of the tide; (4) the duration of onshore winds; (5) the barometric pressure; and (6) the fetch.

Preliminary Results

The pressure gradient. For the four storms studied at the outset, those of November 25, 1950, November 6 to 7, 1953, October 14, 1955, and January 10, 1956, the graphs of meteorological tide versus pressure gradient showed a definite correlation in the first three cases and some scattering in the last case.

The wind velocity. Similar graphs were drawn for the meteorological tide versus the wind. These were to some extent similar to the pressure gradient graphs, but the curve was not so well defined. The wind speeds varied considerably among the various stations in the New York area. It was found that no one station could be classed as having a "representative" wind speed that would correlate well with the meteorological tide in the cases studied.

The phase of the tide. As could be expected, graphs showed that there is a tendency for the meteorological tide to be higher at low tide than at high tide.

Duration of the wind. A graph was made of meteorological tide versus duration of the wind. There was some tendency for larger values of surge

to be associated with longer duration. Results were inconclusive due to lack of data at the time these graphs were made.

Actual barometric pressure. Graphs were made of actual pressure against meteorological tide with pressure gradient plotted for each point in the body of the graph. Although the meteorological tide increased with decreasing pressure, analysis of the graph showed that this might be due to the pressure gradient alone.

Fetch. No graphs were drawn up initially for the fetch.

First Attempts at Establishing Empirical Relationships

Since the meteorological tide was considered a function of several parameters, they were combined linearly in a single equation:

$$\Delta H = f(\Delta P) + F(P) + \psi(T) + \gamma(t)$$

where ΔH = meteorological tide; ΔP = pressure gradient; P = barometric pressure at New York; T = function of the tidal phase; and t = duration of strong winds or gradients.

A solution of the following form gave the best fit for the January 1956 storm:

$$\Delta H = 0.18\ \Delta P - 8.5\ (29.92 - P) + 0.45\ (1\text{-Cos}\ \theta) + 0.17t\ - 3.4$$

where ΔH = meteorological tide in feet; ΔP = pressure gradient/3° latitude in mb.; P = sea level pressure (inches Hg); t = duration of wind hours; θ = angle in degrees corresponding to the tidal cycle; and 0° = high tide counterclockwise to 180° low tide.

It may be seen readily that the pressure effect is directly opposite to what the inverted barometer theory would lead one to expect. The effect of low tide is to add 0.9 feet to the meteorological tide. Steady-state conditions for minimum duration were not considered.

Dimensionless Parameters

The storm of January 7 to 11, 1956, was investigated by means of dimensionless parameters. For $G_1 = \dfrac{W T \Delta P}{P}$ and $G_2 = \dfrac{\Delta H}{WT}$ where W = wind velocity; $T = 6 + t$, where t is the number of hours to the nearest high tide; ΔP = pressure difference per 3° latitude; P = actual sea level pressure at New York (in mb.); and ΔH = meteorological tide (in feet). Two equations were obtained:

$$G_2 = 94{,}700\ G_1^{-1.98}$$

and

$$G_2 = 45\, e^{-0.0135 G_1}$$

Maximum Surge Points

The maximum surge points for each of three storms, those of November 25, 1950, November 7, 1953, and October 14, 1955, were analyzed using dimensionless parameters. The best fit was given by the equation

$$G_2 = 0.0781\, e^{0.158 G_1}$$

where $G_2 = \dfrac{\Delta H}{W(11.67\text{-}t)}$, and $G_1 = \dfrac{W \Delta P}{P}$ $(11.67\text{-}t)$. The parameters are identical to those given in the preceding paragraph. This equation is not satisfactory for several reasons.

(1) For a fictitious storm with extreme but reasonable values of W, ΔP, and P, G_2 can become quite large and ΔH can easily attain values of 30 feet. (2) Using the January 1956 storm as a control storm, the equation when solved for ΔH gave a value of 2.3 feet against an actual reading of 3.7 feet. (3) The forecasting of the maximum surge point also entails forecasting the phase of the tide.

All the above projects had been limited in scope due to the lack of data. When more data had been processed and received, a more ambitious program was instituted. In addition to the simple method for forecasting surge now in use for the New York City area, given at the end of this paper, the following program is now in progress:

(1) Record high and low tides for the past 70 years have been obtained, together with the winds taken from the local records. A statistical study will be made.

(2) Sustained strong northeast, east, southeast, and south winds for the past 20 years are being correlated with the meteorological tides.

(3) The meteorological tides for about 200 days of the year 1953 will be correlated with the average wind speed, the maximum wind speed, the duration, the pressure gradient, and the fetch. These tides will be classified according to direction.

(4) For projects 1 to 3, the dimensionless parameter $\dfrac{gh}{V^2}$ will be plotted against $\dfrac{gt}{V}$. This project was suggested by G. Neumann.

(5) A more detailed analysis of tropical storm surge will be made, especially with regard to resonance and resurgences.

(6) The actual meteorological tide at various stations in the New York area will be compared to the theoretical tide obtained by the Reid and Wilson method.[9]

Latitudinal and Longitudinal Cross Sections of Tidal Heights for Major Hurricanes

Maximum tidal heights for each major hurricane since 1938 were plotted along (1) the north shore of Long Island Sound, (2) the south shore of Long Island Sound, (3) the south shore of Long Island, and (4) the

shore line from northern New Jersey up the Hudson, East, and Harlem Rivers.

Distinction was made between those shore stations directly exposed to the open sea or sound and those in coves and bays. Values were plotted for all the hurricanes, but the data were generally so sparse for some of them (one or two points per coast line) that cross sections could be drawn only for the September 1938, September 1944, and the August 31, 1954 (Carol), hurricanes.

The hurricane path shown on the graph (FIGURE 1) for the hurricane of September 1938 is the one originally given in the *Supplemental Report on Hurricane of September 21, 1938 and its Effect on the Coastal Region,* War Department, Corps of Engineers.[10] This report gave the center as passing five miles west of New Haven, Conn. However, the eye of the hurricane was reported seen at New Haven, and a recent estimate has put the wind center west of this point, and the pressure center over the city itself. The shift of a few miles does not alter significantly the relative over-all high water pattern for Long Island Sound, but the highest tides were reported over the extreme western end

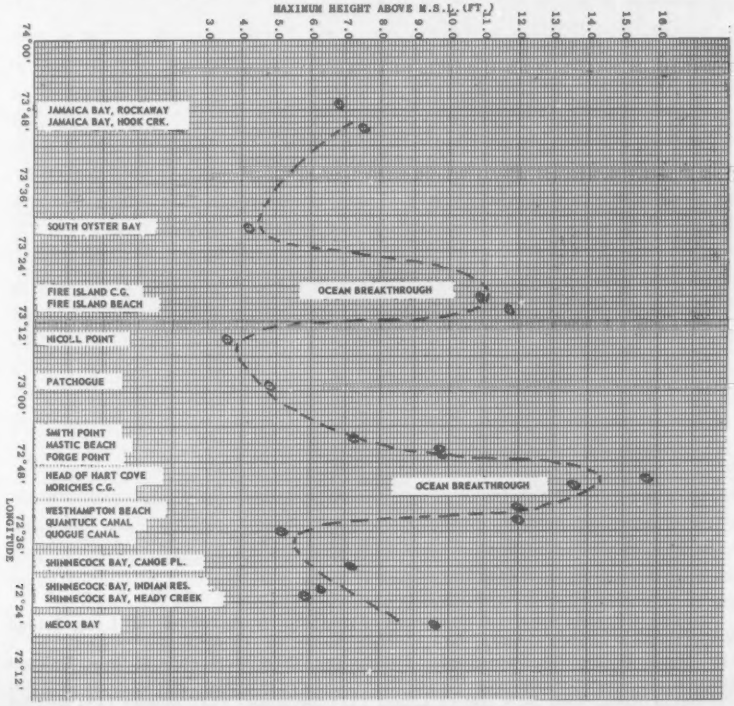

FIGURE 1. Hurricane of September 21, 1938. South shore of Long Island---

of the sound. On the south shore of the sound, the pattern was similar, to some extent, except near Montauk Point, where tides of more than 15 feet were reported. The only explanation that seems reasonable is that a resonance surge built up east of Montauk Point, but was damped out before it moved any distance into the sound (New London tides averaged about 6 feet lower).

On the south shore of Long Island itself (FIGURE 2), the picture is even less clear. The ocean broke through the outer islands in two places,

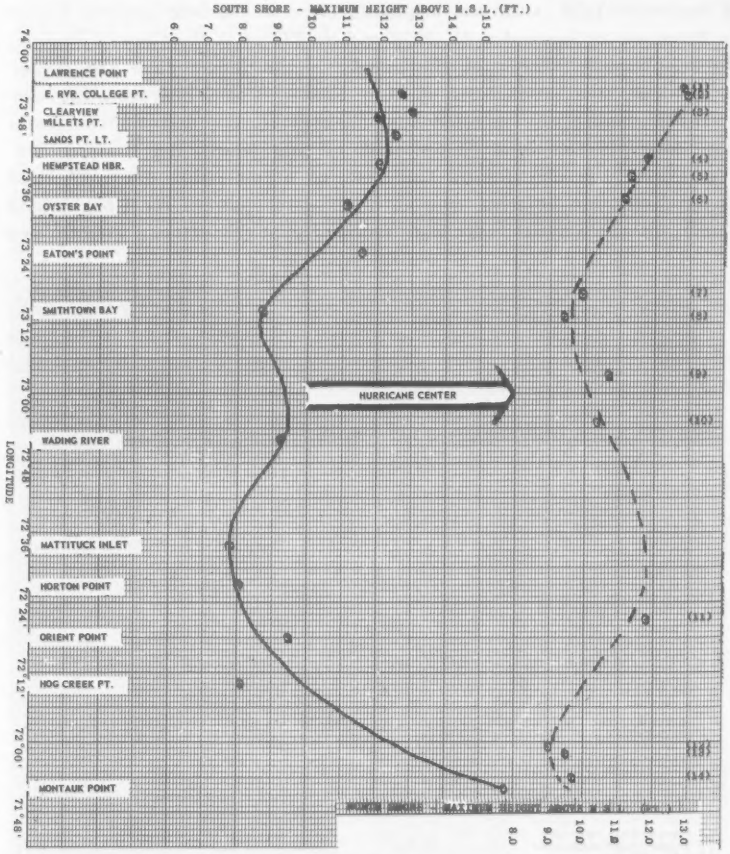

FIGURE 2. Hurricane of September 21, 1938. North shore of Long Island Sound – – – – coves, bays, etc.; South shore of Long Island Sound ——— (1) Hunts Point; (2) Westchester Creek; (3) Throg's Neck; (4) Port Chester Harbor; (5) Greenwich; (6) Stamford; (7) Southport; (8) Black Rock Harbor; (9) Savin Rock; (10) New Haven Light; (11) Saybrook; (12) Noank; (13) Mystic; and (14) Stonington.

one to the left and one to the right of the path (assuming the hurricane moved northward close to the 73rd meridian). The breakthrough on the right caused the highest waters. In both cases, however, stations within the same bay subject to the same breakthrough, where one would expect similar surges, showed a wide variation in high water.

Data were sparse for the September 1944 hurricane. The surge was generally lower in Long Island Sound. In both storms, however, tides were markedly higher in the upper reaches of the East River than in the lower (values not shown in table). This may be due possibly to the piling up of waters from the sound and from the lower East River.

Little data for cross-sectional purposes were available for the 1954 hurricanes except in the case of Hurricane Carol, that of August 31, 1954. Compared to the hurricane of September 1938, Hurricane Carol, with a few isolated exceptions, produced generally lower tides on the north shore of Long Island Sound and higher tides on the south shore of the sound. The surge at Montauk was considerably lower in Carol, although resonance occurred offshore in both cases. This is presumably due to the difference in the paths of the two hurricanes. The fact that Carol passed close to Montauk and was accompanied by a fairly sharp wind shift in that area may have contributed to a rapid dampening out of the resonance factor. On the other hand, in the September 1938 hurricane the path was over the central part of Long Island, and the wind shift was more gradual. The wind action therefore was less effective in reversing the direction of the water transport at Montauk Point.

The effect of wind shifts on water transport in bays, coves, and more landlocked waters is, of course, much more complex and not subject to simple analysis. It has been noted that in some cases, after a windshift to the northwest, the meteorological tide decreases fairly rapidly, while in others, it remains stationary or decreases slowly. In the latter cases, there is generally a very slow-moving or stationary surface pressure trough offshore that apparently blocks any large mass transport of water away from the coast.

Tide Deviation Correlation between the Various Tide Gauge Stations in the New York District

When tides run above or below normal at the Battery, the southern point of the island of Manhattan, there is obviously a strong tendency for the tides to act in the same direction in the rest of the New York district. The exceptions occur notably where a front exists between the two stations or where the pressure pattern is such as to cause a deviation at one station and not at another. The latter exceptions seldom occur when the two stations are close together and follow the same tidal regime (the Battery and Sandy Hook, N.J.), but do take place somewhat more frequently when the stations are farther apart and follow different tidal regimes (the Battery and New London, Conn.).

Before and during storms that cause deviations from the predicted tides, there is a continuous service demand for information. They can be divided into two types of questions: (1) How much is the tide running above normal now in Area X? (2) How much do you expect tides to run above normal in Area X tonight? Area X in most cases was an area from which no direct tidal information could be obtained. Therefore, it was necessary, during the early stages of the surge forecast program, to give estimates based on rather sparse data. To fill this gap in the service requirements, the following simple method was devised:

Using the data for 1953, a linear regression line was computed for the deviation from predicted tide at two stations. For example, maximum daily deviation (positive or negative) at the Battery was plotted against the maximum daily deviation at Montauk Point. Deviations of absolute values of less than one-half foot for either station were not included, since there was some doubt as to their usefulness and significance. This eliminated a considerable number of cases. The equations and correlations shown in TABLE 2 were found for the year 1953.

TABLE 2

x	*y*	*Cases*	*Equation*	r_{xy}
The Battery	Sandy Hook	194	$y = 0.1 + 0.93x$	0.962
The Battery	Willets Point	208	$y = 0.1 + 1.15x$	0.961
The Battery	Montauk Point	164	$y = 0.1 + 0.75x$	0.955
The Battery	New London	131	$y = 0.2 + 0.78x$	0.955
Willets Point	New London	171	$y = 0.2 + 0.62x$	0.943
Montauk Point	New London	164	$y = 0.2 + 0.98x$	0.972
Willets Point	Montauk Point	242	$y = 0.1 + 0.62x$	0.935

The average number of cases studied was 182, or a period of approximately one-half year. The correlation is very good in all the above instances, but it must be remembered that only those cases in which both stations registered deviations of one-half foot or more from predicted tide were chosen. In a few cases, the exceptions mentioned under the first paragraph of this section were a controlling factor. For the remaining days of the year, no data were received.

This simple type of graph is quite useful as both an observational and forecasting tool. Experience has shown that the ΔP versus Meteorological Tide graph, worked up at this station, proved itself quite valuable and fairly accurate during the storms last autumn. Use of the same graph for the prognostic charts resulted in good forecast values. By the use of the ΔP versus Meteorological Tide graph for the Battery, in combination with the correlation graphs mentioned above, it was possible to make better forecasts for the other stations in the area. We were also able to

state, with a fair degree of confidence, how high the tides were running in a given area.

The Meteorological Tide versus the Pressure Gradient

A graph of the meteorological tide versus the pressure gradient, based on the data from 5 major storms, was worked up.

The pressure gradient is measured per 3 degrees of latitude. The graph is then entered and the meteorological tide is read off. This method can also be used on a prognostic chart and allows us to approximate the meteorological tide for the verification time of that chart.

In actual practice during the past ten months, good forecasts of expected surges have been made by the use of this method. It is possible, however, that in view of the fact that such parameters as duration and fetch are not specifically taken into account, that some of this is due to chance. As yet, it has not been tested on a tropical storm, since none approached the New York area last year. As in the case of all weather forecasts, this method depends primarily on a good prognostic map. It is hoped that, by the next hurricane season, a more refined method for forecasting surges will be available.

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Elected May 15, 1957

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The legend of FIGURE 2 should have appeared under FIGURE 3, and the legend of FIGURE 3 under FIGURE 2.